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### A STUDY OF MYOTONIA, WITH SPECIAL REFERENCE TO PARAMYOTONIA.

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#### PART I: MYOTONIA IN HUMAN SUBJECTS.

##### Definition.

MYOTONIA is a painless persistent contraction of a muscle occurring after voluntary, mechanical, electrical and occasionally reflex stimulation. It occurs typically in *myotonia congenita* and also forms part of the symptom complex of *myotonia atrophica*. In addition there are two rare variations of the myotonic syndrome, *paramyotonia* and *myotonia congenita intermittens*. All these diseases have a strong familial incidence.

##### Nomenclature.

At the outset it is necessary to clarify the nomenclature of these diseases. Maas and Paterson<sup>(34)</sup> consider *myotonia congenita* and *myotonia atrophica* to be the same disease, which they have designated *dystrophia myotonica*, a term previously applied only to *myotonia atrophica*. We intend to separate the myotonic syndrome into *myotonia congenita*, *myotonia atrophica*, *paramyotonia* and *myotonia congenita intermittens*.

##### Historical Review.

The disease was first described under the title of "tonic spasms of voluntary muscles in consequence of an inherited disposition", by J. Thomsen,<sup>(37)</sup> a Danish physician, who himself suffered from it. He performed experiments on his son, who had been subjected to unjust treatment as a

result of the malady while serving in the military forces. The observations were published with the object of preventing similar injustice to other sufferers. Thomsen drew attention to the cramp which occurred on voluntary contraction, its onset in childhood, its distribution through the voluntary muscles, and its hereditary nature, as demonstrated by its occurrence through several generations of his own family. Strümpell<sup>(38)</sup> suggested the name *myotonia congenita*. Erb<sup>(39)</sup> accepted this term and the muscular abnormality became known as myotonia. Erb's work was of great importance, for not only did he establish the clinical picture, but he also gave detailed pathological findings and a description of the electrical responses typical of the disease and known as the myotonic reaction. As a result, abnormal types were described, such as *paramyotonia* and *myotonia congenita intermittens*.

In 1900 Hoffmann<sup>(40)</sup> described atrophy in association with myotonia. Batten and Gibb<sup>(41)</sup> and Stienert<sup>(42)</sup> independently showed that the atrophy was limited to a special distribution. In 1912 Curschman<sup>(43)</sup> recognized the extramuscular associations of the disease now known as *myotonia atrophica*, first identified by Rossolimo<sup>(44)</sup> in 1901.

Important contributions were made by various authors<sup>(45)</sup> on abnormal types of the disease, on the creatinine metabolism<sup>(46)</sup> and on the genetics.<sup>(48)</sup> Jelliffe and Ziegler<sup>(49)</sup> summarized the literature. Fleetwood<sup>(50)</sup> published the first Australian case in 1893.

Interest was awakened in abnormal muscular pathology by the publication of the work of Dale<sup>(51)(52)(53)</sup> and his school on neuromuscular transmission. The discovery that *myasthenia gravis* could be relieved by "Prostigmin"<sup>(54)</sup> resulted in the application of the same physiological principles to myotonia.

However, studies in choline esterase estimations in the disease<sup>(77)(84)(85)</sup> gave conflicting results, and it appeared that Dale's concepts could not be applied to explain the abnormal pathology. The description by Wolf<sup>(90)</sup> of the effectiveness of quinine in treatment led to the publication of further articles. "Prostigmin" and quinine were shown to be antagonistic in myotonia and myasthenia

<sup>1</sup> An expansion of this paper was presented as an M.D. thesis at the University of Adelaide by J. S. Covernton, 1946.

<sup>2</sup> The work was assisted by grants from the National Health and Medical Research Council.

respectively.<sup>(44)</sup><sup>(46)</sup> The mode of action of quinine was elucidated<sup>(47)</sup><sup>(48)</sup> and the results were applied clinically.<sup>(48)</sup><sup>(49)</sup><sup>(50)</sup><sup>(51)</sup><sup>(52)</sup>

In 1938<sup>(53)</sup> it was found that a strain of goats resident in the United States of America were subject to an hereditary disease almost identical with myotonia. From extensive electromyographical investigations in these animals, Brown and Harvey<sup>(51)</sup><sup>(52)</sup> concluded that the site of the abnormality was probably in the muscles themselves, rather than in the nervous system. On the other hand, other observers<sup>(51)</sup><sup>(50)</sup> were of the opinion that the defect was the result of central influences of a reflex nature acting on the abnormal muscles.

Considerable work has been done on the hereditary aspects of myotonia and on its natural history.<sup>(53)</sup><sup>(54)</sup><sup>(55)</sup> The conflicting opinions will be the subject of further discussion.

#### Clinical Picture.

As has previously been stated, the syndromes characterized by myotonia are as follows: (i) *myotonia congenita*, (ii) *myotonia atrophica*, (iii) paramyotonia, (iv) *myotonia congenita intermittens*. A fifth type, *myotonia acquisita*, has been described,<sup>(56)</sup> but it is of doubtful existence.

With the exception of *myotonia acquisita*, all these syndromes have a strong familial tendency. However, the actual mode of inheritance is still a matter of controversy. Working on *myotonia atrophica*, Ravin and Waring<sup>(56)</sup> considered that it was transmitted by a single dominant factor modified by progressive inheritance. On the other hand, Nissen, investigating *myotonia congenita*, believed that it was transmitted by a pure Mendelian dominant inheritance. Other workers<sup>(53)</sup> contend that the complexes are variants of the one disease and that the inheritance is multifactorial—that is, one factor transmits myotonia, one cataract *et cetera*. Many of these authors' statements are open to criticism. On the basis of the statistical data available in this inquiry, we are of the opinion that the mode of inheritance is dominant, and the genealogical trees of the families studied bear this out (genealogical tables, Figures I and II).

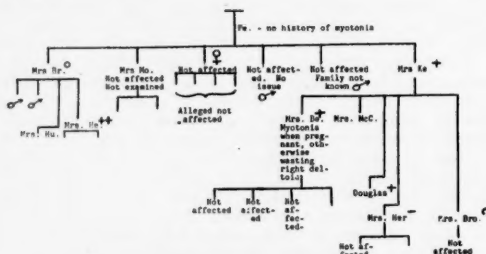


FIGURE I.

Genealogical table, Family I: "+" myotonic, examined; "O" myotonic, not examined; "-" not affected; "Alleged not affected" alleged myotonic when pregnant, but examined and found clinically well; "Alleged not affected" alleged myotonic when pregnant, not examined; no distinguishing mark, not affected with myotonia.

We believe that a study of the syndromes will show that a close relationship exists between them. Many authors<sup>(75)</sup><sup>(76)</sup><sup>(77)</sup><sup>(78)</sup><sup>(79)</sup> have shown that atrophy may occur in *myotonia congenita*, and that certain members of the families exhibiting *myotonia congenita* may exhibit paramyotonia.<sup>(55)</sup><sup>(58)</sup><sup>(59)</sup> One of our subjects, Mrs. Be., exhibits muscular atrophy, while all her family show true *myotonia congenita*. An attempt will be made to clarify the clinical picture of the several diseases with additions and modifications noted as a result of personal observations in the cases described.

#### *Myotonia Congenita: Family I.*

The disease is familial (see Figure I). The patients complain of muscular stiffness coming on early in life. The stiffness follows voluntary movement and takes the form of difficulty in relaxation of muscles. Repetition diminishes the stiffness, which is painless; it is of general

distribution, but is most apparent in the calves and thighs and in the hands and arms. Frequently it is present in the neck and occasionally in the jaws.

In Family I the eyes are never affected, the tongue sometimes, and speech seldom. In certain circumstances generalized stiffness occurs. The most potent cause of this phenomenon is the combination of fright and sudden movement. All of our patients have had these attacks, which lead to sudden falls without loss of consciousness. Because of the stiffness it may take minutes before they can rise. Unlike most other sufferers from this disease, in this family cold did not induce the stiffness. D.K. was examined by myographic methods before and after immersion of his arm in cold water at 8° C. for fifteen minutes. No increase in muscular stiffness was observed.

Sudden forceful movements are more likely to cause stiffness than slow gentle movements. Fright or nervousness always makes the patient worse. Pregnancy has a remarkable influence on the condition in this family. Griffith<sup>(60)</sup> has stated that pregnancy relieves the disability. In the family under discussion there are five married women. Three show evidence of myotonia, but only one of these, Mrs. K., is constantly affected. In all of her eight pregnancies she has become worse usually at about the sixth week. The stiffness is severe and occurs more frequently and in the same distribution. The effect lasts throughout pregnancy and ceases when gestation ends. Three of eight pregnancies have terminated prematurely, and the aggravation of symptoms ended with the extrusion of the ovum. In the five normal pregnancies labour was not affected. Lactation had no effect. All other affected females have been worse during all their pregnancies. Mrs. Br. has been examined in both the pregnant and the non-pregnant state, and myotonic symptoms cannot be detected in the latter.

Examination of the patients reveals generalized muscular hypertrophy. One subject, D.K., had the classical Herculean development. Mrs. Be. exhibits wasting of the anterior portion of the right deltoid muscle.

The muscles are firm. Forceful closing of the hand results in delayed opening, which decreases with repetition. On the fifth closure the rate of relaxation is normal.

Myotatic irritability is pronounced. If the arm is placed on the table with the elbow flexed and the wrist resting in a position of pronation, and if the muscles on the lateral aspect of the forearm are struck, flexion of the wrist and supination of the forearm occur. The movement is quick in contraction and slow in relaxation. If the deltoid is struck, a furrow develops in the vicinity of the point struck which slowly subsides from the periphery. Repetition of blows of approximately equal intensity results in a progressive diminution in the relaxation time. The rate of relaxation is roughly proportional to the strength of the blow.

Electrical examination of these patients was refused. The classical electrical reaction in myotonia is as follows. The muscular response to faradic and galvanic stimulation lasts longer than normal, and is more obvious the stronger the current used. On prolonged application of galvanism, slow wave-like contractions pass from cathode to anode.

Muscular power was shown to be less than normal in all but D.K. The tendon reflexes in these patients show no delay in relaxation. No sensory changes were noted. Various myotonic manifestations have been described in association with eye movements.<sup>(61)</sup> No abnormalities of the eye movements were found in this family. One constant finding was present in both families, to which no reference has been made by other authors. The response of the pupils to light was a fluctuating response (hippus), while the response to accommodation was normal. No other abnormalities were detected in the central nervous system. The skin over the extremities was bluish and showed evidence of vasomotor instability. Dermatographism was present in three cases. Bruising of the skin was common in two cases; small ecchymoses of more than average numbers were observed in two out of three cases on several occasions.

With regard to the mental condition of these patients, it may be stated that members of the family display a peculiar reluctance to be examined and helped. Similar findings have been reported previously,<sup>(62)</sup> and some patients go to great lengths to conceal their disability. Their intelligence is good. All are constantly employed. There is no family history of mental retardation or insanity, and no evidence of progressive social degradation such as has been reported elsewhere.

#### *Myotonia Atrophica.*

Myotonia has been estimated to be accompanied by atrophy in 9% to 11% of cases.<sup>(63)</sup> Some investigators have urged<sup>(64)</sup><sup>(65)</sup> that it is a not uncommon disease and

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that cases are often diagnosed as muscular atrophy from other causes. For the most part the onset of atrophy occurs in middle life, but cases have been reported even in the first decade.<sup>(10)(14)</sup> Atrophy may be preceded by loss of vibration sense.<sup>(14)</sup>

The atrophied muscles usually include the facial muscles, the sterno-cleido-mastoids, the muscles of the forearm, the extensor muscles of the leg and the dorsiflexors of the foot. The affected muscles show no fibrillary tremors. Atrophy may affect muscles showing myotonia or others. As a consequence of the atrophy of the facial and temporal muscles the following are observed: ptosis, hollow cheeks and dimples, and a straight mouth with a turned-down smile resulting in the appearance known as the myopathic facies. As the atrophy advances the myotonia tends to recede and eventually may be difficult to discover; it should be sought for in the hand grasp and the jaw. The myotonia differs in no way from that associated with *myotonia congenita*. Myotonic electrical responses together with myotatic irritability are obtainable in unatrophied muscles. Accompanying the myotonia and atrophy are other dystrophic symptoms. In a large percentage of cases<sup>(10)(14)</sup> cataract is present when searched for by slit-lamp microscopy. Rarely, cases have been reported in which the muscles are affected without the presence of cataract. Cataract may also occur as an isolated symptom in a myotonic family.<sup>(10)(16)</sup>

The cataract produces small irregular opacities in the cortex and takes ten to twenty years to mature; it then differs in no way from other mature cataracts.

**Testicular Atrophy.**—Testicular atrophy is common in the fully developed disease and leads to impotence and infertility. In the female menstrual irregularity is the rule, and pregnancy is unusual in women aged over thirty years who suffer from this disease. Baldness is the rule in men. The thyroid gland is often enlarged. The basal metabolic rate is low.<sup>(16)</sup>

**Mental Changes.**—Pronounced muscular wasting is often associated with mental changes characterized by mild grandiosity and morbid cheerfulness. On the other hand, Ravin and Waring consider that any changes in temperament are the result of the reaction to bodily defects rather than otherwise. Thomsen's original family description showed many instances of mental instability, and there is reason to think that the incidence of mental defects is higher in affected than in unaffected families. Mild forms of the disease are certainly compatible with intellectual health.

#### Paramyotonia.

The third group of cases comprise those of paramyotonia and *myotonia congenita intermittens* (Figure II). As the former is the main part of the study undertaken on the myotonic diseases, it will be dealt with in some detail.

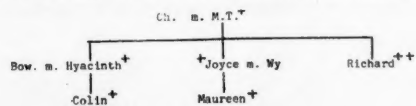


FIGURE II.

Genealogical table, case protocols, Family II; "m.", myotonic; "++", suspected myotonic, serving with the Australian Imperial Force in New Guinea.

The term "paramyotonia" was first used by Eulenberg and Melchett in 1885.<sup>(17)</sup> They applied it to families in which attacks of temporary stiffness occurred on voluntary movement while the patient was under the influence of cold. The condition had persisted through a generation and was detectable at birth. The attack lasted from one to two hours, and at its height the patient used to fall and be unable to rise without assistance. Although no paralysis was present, a sensation of weakness remained in the muscles for twenty-four hours. The disability occurred only in cold weather. Electrical examination revealed no abnormally persistent contraction. Delprat<sup>(18)</sup> described a family in which spasms similar to those reported by Eulenberg were present, the members of the family being affected through several generations. In two members of

the family after their fifteenth year the spasms changed until they presented the appearance of *myotonia congenita*. Von Solder<sup>(19)</sup> reported a case in which the stiffness was much worse in cold weather. In summer slow easy movements took place with no disturbance. If quick forceful movements were made profound disturbance occurred. The first contraction occurred with the minimum of trouble. If the movement was repeated, the muscle remained contracted for several seconds. On further repetition, increasing delay in relaxation accompanied by a sense of weakness occurred and in the end no contractions were possible. Such attacks were aggravated by cold weather. Exposure to cold led to a substantial reduction of muscular strength. Under these conditions the muscular stiffness set in after the first contraction and increased to absolute rigidity. Warmth diminished the stiffness. Mechanical irritability was increased and investigation of electrical responses showed a persistence of contraction. Further cases occurred in the same family. Schott<sup>(17)</sup> has also described cases. Hlawaczek<sup>(20)</sup> observed a case in which the same phenomena occurred in conjunction with myotonia. Complete stoppage of movement was present only in those muscles exposed directly to cold.

These cases have certain common differences from *myotonia congenita*; they are as follows: (i) the onset of stiffness under the influence of cold; (ii) the appearance of weakness or paresis following severe attacks of stiffness; (iii) in some cases the absence of myotatic irritability together with atypical electrical responses; (iv) increase of symptoms on repetition of movement, instead of improvement.

On the other hand, the family described by Delprat<sup>(18)</sup> showed the close relationship between the two diseases. Pelz<sup>(21)</sup> has recorded atypical manifestations of the disease. Various authors have observed cases in which, under the influence of cold, certain muscles manifested spasm and delayed relaxation. Martius and Hausemann,<sup>(22)</sup> however, described a unique case characterized by attacks of muscular stiffness appearing only in cold weather. At most, the attack lasted a few hours. The spasms were not accompanied by weakness, nor did repetition of the movement result in improvement. Previous generations had suffered from *myotonia congenita* in pure form. This case would seem to be a link between paramyotonia and *myotonia congenita*; the condition was called *myotonia congenita intermittens*. Other cases have been recorded<sup>(23)</sup> in which weakness, unaccompanied by spasm, occurred on intended movement which if repeated gave way to increasing functional capacity. The electrical reactions were typical of myotonia.

From an analysis of our findings together with study of the literature it would appear that the cases in Family II are classical examples of paramyotonia, embracing all phases of the symptom complex. In conformity with this view the subjects present weakness, fail to improve on repetition of the movement, and have a strong familial tendency to the condition. A clinical description of paramyotonia will be given, based on observation of a family suffering from the disease.

**Clinical Picture of Paramyotonia.**—The patients complain of stiffness and difficulty of muscular relaxation occurring on voluntary movement, which may occur in any voluntary muscle. It is shown best in the hand, which on being forcefully closed may remain closed for several seconds. The stiffness is painless, but when severe is associated with a feeling of tension in the affected part. Minor degrees of stiffness are always present, and can be lessened by slight movement. However, if at certain times the patient is called upon to perform a sudden movement or to repeat a given movement, more severe stiffness is likely to supervene. This is often the result of movement in cold weather. The most important factor is cold; but fatigue, hunger, menstruation and pregnancy are also likely to increase the severity of the attacks.

The distribution of the stiffness may be general. If so, the arms are involved more than the legs; but usually the latter take longer to recover. Associated with severe attacks is a feeling of weakness described as uselessness. Continuation of forceful movements invariably increases the



stiffness and weakness, ultimately leading to complete functional incapacity. At these times it is necessary for the patients to move slowly about if they wish to move at all. This is possible, though difficult at the height of an attack. Warmth relieves the condition. At such times movements can be performed if they are not too forceful. Attacks may occur daily, according to the temperature. Their duration is twelve hours or less and it may sometimes be shortened by rest in bed. Rest without warmth is unsatisfactory. Falls without loss of consciousness are frequent during the attack; they are usually the result of tripping. The stiffness already present is aggravated, the patient becomes "weak" and "goes in the ankles" and falls. He may remain helpless for half an hour, and always has to be assisted to his feet.

In the intervals between the attacks, some residual stiffness is constantly present. It does not seriously incommode the patient and may be reduced with exercise.

The condition seems to increase in severity with age. The residual stiffness is more evident and the number and severity of the attacks are greater. Two patients have been observed throughout pregnancy. In both instances the number and severity of the attacks increased. The process of labour was uncomplicated and assistance was limited to the end of the second stage.

There is always evidence of strong family inheritance.

All members of Family II, which has been observed for three generations, are affected.

We noticed that sweating was more pronounced than usual in three out of five cases. Bruising was more frequent than usual, and ecchymoses were present in four out of five cases.

Physical examination disclosed muscular hypertrophy, which contrasted with the long myopathic type of face and a mouth with a flat smile. The eyes in all cases showed evidence of unstable reaction to light. The response to accommodation was brisk and maintained. No evidence of cataract was found by Dr. R. M. Glynn. Except for the facial appearance there was no evidence of dystrophic manifestations in any of the five patients. One patient, W.T., aged sixty-five years, manifested arterial disease causing symptoms of coronary occlusion, considered to be unrelated to his muscular condition. Examination revealed the muscular hypertrophy to be general. The hands were held in a position of flexion at the wrists and metacarpal joints with the thumb in opposition. Active movement of the wrist was limited by the position of the fingers. Active hyperextension of the wrist with the fingers extended was limited to 30°. Passive movement could not be performed through a full range of movement. In all five cases, if the fingers were flexed, movements of the wrist were of normal range. Gentle closure of the hand caused no serious delay in relaxation. Repetition of gentle closure might be performed, but a sense of tightness developed. Prolonged forcible closure of the hand resulted in a twenty second delay in opening, the hand being held in an exaggerated condition of flexion as described above. Repeated forcible closure caused increasing spasm with each contraction,

until ultimately the hand was powerless and held in a position of flexion. Afterwards the hand slowly opened, the opening sometimes taking as long as five minutes to complete. Both flexor and extensor muscles felt hard and knotty.

Immersion of the arm in cold water at 8° C. resulted in an exaggeration of the effects described. These phenomena are shown on the myograms (Figures VIII, IX, X and XI) and will be more fully discussed later.

The response to the mechanical stimulus of percussion was increased. In the line of the fibres of the muscle struck a furrow appeared, which persisted for a variable time and subsided from the periphery to the centre. Repeated blows caused no change in the rate of relaxation.

The electrical responses of the muscle were in accord with Erb's classical description. Delay in relaxation to both faradic and galvanic stimulation was observed. Contractions were brisk. The delay was increased with an increase in strength of the current. The delay lasted five to ten seconds after galvanic stimulation. When the galvanic current had been flowing for a few seconds, wave-like contractions passed from cathode to anode. With the reversal of the direction of the current, the direction of the waves was reversed.

#### Characteristics of the Myotonic Diseases.

The characteristics of the myotonic diseases are shown in Table I.

#### Myotonia Acquisita.

Two more alleged types of myotonia remain to be considered.

The first is *myotonia acquisita*. Talma<sup>(80)</sup> maintained that myotonia could be acquired. Although most authorities disagreed, Krabbe<sup>(81)</sup> revived the idea and considered that myotonia might result from intoxications similar to those responsible for peripheral neuritis. Critical examination of his cases led Ravin<sup>(82)</sup> to conclude that if strict criteria for diagnosis were applied, there was no such disease.

#### Thyreoid Diseases.

A myotonia-like muscle disturbance has been demonstrated clinically and electrically in association with myxœdema.<sup>(87) (88) (79) (14)</sup> Other cases of myxœdema have been associated with painful cramps and tonic spasms.<sup>(89) (90)</sup>

The diagnosis of myotonia is open to doubt in these cases for the following reasons: (i) thyreoid extract has no effect on the myotonic symptoms of *myotonia atrophica* even when the condition is accompanied by a lowered basal metabolic rate; (ii) electromyographic tracings taken<sup>(91)</sup> are not typical; (iii) the electrical reactions are also not typical in most cases.

The two signs found in common with myotonia are muscular hypertrophy and myotatic irritability, both of which disappear on the exhibition of thyreoid extract in suitable doses. It seems extremely doubtful whether true myotonia exists in association with thyreoid deficiency.

TABLE I.  
(After Ravin.)

| Author.                | Diagnosis.                               | Effect of Cold.    | Presence of Paresis.      | Effect of Repetition of Movement. | Electrical Reaction.                        | Mechanical Reaction.              | Occurrence in Attacks.                           | Occurrence on any Intended Movement. | Effect of Strength of Contraction. |
|------------------------|--|--------------------|---------------------------|-----------------------------------|---|-----------------------------------|--|--------------------------------------|------------------------------------|
| Thomson <i>et alii</i> | <i>Myotonia congenita</i> .              | Doubtful (J.S.C.). | Doubtful.                 | Improves.                         | Myotonic reaction.                          | Present.                          | No.  | Yes.                                 | No effect.                         |
| Hoffman <i>et alii</i> | <i>Myotonia atrophica</i> .              | Doubtful (J.S.C.). | Doubtful.                 | Improves.                         | Myotonic reaction in non-paralysed muscles. | Present in non-paralysed muscles. | No.  | Yes.                                 | No effect.                         |
| Eulenberg              | Paramyotonia.                            | Worsens.           | Severe, almost paralysis. | Worsens.                          | Myotonic reaction absent.                   | Absent.                           | Yes.   | No.                                  | Worsens if strong.                 |
| V. Schulaer            | Paramyotonia.                            | Worsens.           | Severe.                   | Worsens.                          | Myotonic reaction present.                  | Present.                          | Some residual myotonia, but exacerbations occur. | No.                                  | Worsens if strong.                 |
| Schoff                 |  |                    |                           |                                   |   |                                   |  |                                      |                                    |
| Rich                   |  |                    |                           |                                   |   |                                   |  |                                      |                                    |
| Martius and Hansemann. | <i>Myotonia congenita intermittens</i> . | Worsens.           | Absent.                   | Worsens.                          | Myotonic reaction present.                  | Present.                          | Attacks prompted by cold.                        | No.                                  | No effect.                         |
| Stein                  | <i>Myotonia congenita</i> .              | ?                  | Most prominent.           | Improves.                         | Myotonic reaction present.                  | Present.                          | No.  | Yes.                                 | No effect.                         |
| Weichman               | Myotonia.                                | ?                  | Absent.                   | Worsens.                          | Present.                                    | Present.                          | No.  | Yes.                                 | Occurs only on strong movements.   |
| Wichman                |  |                    |                           |                                   |   |                                   |  |                                      |                                    |
| J.S.C., Cases I to V.  | Paramyotonia.                            | Worsens.           | Present.                  | Worsens.                          | Present.                                    | Present.                          | Yes, in association with cold.                   | Yes.                                 | Worsens if forceful.               |



### Pathology of Myotonia Congenita.

The histological picture of muscle in *myotonia congenita* is variable. Erb<sup>(42)</sup> found that in its complete form the muscles were hard to touch. The colour was not distinctive; in some cases individual fibres of the muscle could be seen. Microscopically, the size of the individual muscle fibres was 25% larger than normal. In cross-sections the fibres were seen to be rounder, and they exhibited a lack of normal cross-striation. In longitudinal sections they were seen to be curved. Vacuoles were present in a proportion of fibres. There was an increase in nuclei, which were located next to the sarcolemmal sheath. In general, these findings have been confirmed by the majority of workers.<sup>(72) (40) (45) (70)</sup> The essential features are the increase of nuclei and the increased size of the fibres. However, histological changes may be slight or absent. Schiefferdecker<sup>(72)</sup> and Schultze<sup>(70)</sup> considered that they might be less pronounced in the earlier stages of the disease. Biopsies were taken from two patients suffering from paramyotonia. Dr. J. B. Thiersch reported that the specimen from the younger subject, K.B., was normal. In the specimen from the older subject, W.T., irregularity in the size of the fibres was seen. Some were decreased and shrunken; in others circumscribed swelling was present with apparent degeneration of the contractile substance, which stained blue with hæmatoxylin. There was an increase of nuclei of the *perimysium internum*.

In *myotonia atrophica* similar changes occur and are roughly proportional to the wasting present. The nuclei are gathered in chains inside the fibre, which ultimately becomes rounded and disintegrated.

The changes noted in W.T. are similar to those of *myotonia atrophica*. On the other hand, in H.B. no lesions were detected. We gained the following impressions: (i) the histological changes are proportional to the severity and duration of the disease; (ii) when present, the changes are similar to those found in *myotonia atrophica* and *myotonia congenita*.

Two autopsies have been performed in cases of myotonia. In one<sup>(43)</sup> the findings are valueless, because no examination was made of the central or peripheral nervous system. In the second,<sup>(40)</sup> some atrophy of cells in the *corpus striatum*, the hypothalamic nuclei, the *substantia nigra*, the *formatio reticularis* and the dentate nucleus was described. In discussing these findings, Kinnier Wilson<sup>(40)</sup> does not accept them unconditionally, but suggests that their abiotrophic nature may be in keeping with the hereditary nature of the disease.

### Diagnosis.

Myotonia may be distinguished from other abnormal muscular contractions if its characteristics are remembered. They are the following: (i) the presence of painless tonic contraction followed by slow relaxation on voluntary mechanical or electrical stimulation of voluntary muscles; (ii) the universal familial tendency; and (iii) in most cases the tendency to relief on repetition of the movement.

Such contractions are unlike the spasms of tetany, which are confined to the hands and feet, are painful, are induced by slight mechanical or emotional stimuli, and are associated with gross metabolic disturbances.

Another abnormal type of contraction to be distinguished is that produced by the habitual use of one set of muscles in constant repetition of movement. The spasm is characterized by local pain and spasm in the muscles concerned, by loss of volitional control of range and movement of muscles, and finally by weakness of movement. It should be emphasized that these symptoms, though produced voluntarily, occur only during the performance of the particular repetitive movement.

On the other hand, the myotonic patient produces his symptoms on any movement.

With regard to the syndromes themselves, *myotonia congenita* may be distinguished by the muscular hypertrophy, the familial incidence and the typical spasm. It has been confused with hypertrophic muscular dystrophy, but in this condition muscular weakness accompanied by large flabby muscles is the salient feature. Moreover, the histological picture is different.

In *myotonia atrophica* the myotonia has often to be sought for assiduously, as it may be confined to the small muscles of the hand. If it is not recognized, it may be designated "progressive muscular atrophy". When the definite distribution of the atrophy is found, and particularly the myopathic facies, the diagnosis is not in doubt. The concomitant dystrophic symptoms appear relatively early, and cataract may be recognized if slit-lamp microscopy is undertaken.

Paramyotonia may be distinguished by the appearance of gross myotonic symptoms under the influence of cold. The other salient features are the presence of weakness in association with spasm and the tendency for increase of symptoms with repetition.

### Prognosis and Course.

By itself myotonia does not tend to shorten life. The generalized spasms which are associated with it have in one recorded instance resulted in death from head injury. The prognosis varies with the symptom complex manifesting the myotonia.

### Myotonia Congenita.

Persons suffering from *myotonia congenita* have a normal expectation of life. The disability has been noticed in a suckling baby,<sup>(40)</sup> and it persists throughout life. Remissions and relapse are said to occur. In most cases the condition becomes more obvious at puberty and thereafter continues unchanged. Death is not due to the myotonia.

### Myotonia Atrophica.

Mild forms of *myotonia atrophica* are slowly progressive and are compatible with a useful existence for many years. More severe forms are rapidly progressive and are often accompanied by mental defect. Such patients usually die at ages ranging from forty to fifty years. Death is often due to intercurrent infection. The degenerative character of the disease may affect subsequent generations, and there is a tendency for them to have the disease in a more severe form. A tendency towards progressive degradation of social status<sup>(40)</sup> has also been recorded.

### Paramyotonia.

There is no record of prognosis in paramyotonia. Members of the family here described develop the disease early. In one case it was recognized in infancy. The condition *per se* does not appear to affect life. There is a tendency for the disability to increase with age. The patient W.T., aged sixty-five years, is the most severely affected in the series.

### Treatment.

#### Quinine.

Until the discovery of quinine<sup>(40)</sup> as a method of treatment, no effective remedy was known; but it was suggested that sufferers should avoid excessive exercise and exposure to cold. Various drugs had been recommended. Erb<sup>(22)</sup> mentions curare, but gives no specific method of administration. He also notes the beneficial effect of alcohol, a finding confirmed by others.<sup>(43) (45) (70)</sup> The benefit obtained<sup>(70)</sup> is proportional to the amount of alcohol taken.

The intravenous administration of 0.75 gramme of quinine hydrochloride abolished in the space of ten minutes all myotonic phenomena for twenty hours; quinine was also effective when given by mouth. Harvey<sup>(43)</sup> believes that quinine has a curare-like action, thereby lessening the effectiveness of the neuro-muscular transmitter, and, further, by its action of diminishing the refractory period of muscle, nullifying the tendency of myotonic muscle to respond repetitively to any stimulus. Given by mouth in doses of 0.5 gramme three times a day, the drug is effective in maintaining the patient symptom-free. Quinine is satisfactory both in *myotonia congenita* and in *myotonia atrophica*. The results in paramyotonia are less conclusive. One patient exhibited an idiosyncrasy to the drug, and myograms taken after its administration showed no improvement. Another child was given 0.24 gramme three times a day for six weeks without benefit. "Syntropan" (Hoffmann-La Roche) has a beneficial effect in paramyotonia; injected in a dose of one millilitre, it alleviates

the severe muscle spasms resulting from the injection of 0.5 millilitre of "Prostigmin". Previous injection of one millilitre of "Syntropan" prevents the development of severe muscular disturbance after immersion in cold water. Clinical trials have not yet been made on account of shortage of the drug, and its use has been restricted to experimental work. At a future date we hope to publish results of clinical trials in all myotonic diseases.

The shortage of quinine has also restricted its use in clinical trials, thus rendering it impossible to pronounce at this juncture on the effectiveness of quinine and "Syntropan" on paramyotonia.

Alcohol was of no benefit in our cases of paramyotonia.

## PART II: MYOTONIA IN ANIMALS.

True *myotonia congenita* can occur in goats.<sup>(85) (47) (11)</sup> These animals have been used to elucidate the nature of the disease and its response to drugs.

### Stringhalt.

The occurrence of myotonia in goats suggested to us that stringhalt, a disease of horses, might have certain features in common with paramyotonia.

We found that in stringhalt there was a strong familial tendency, although from our investigations of the records of a stud we were unable to determine the exact mode of inheritance. The manifestations of stringhalt are entirely muscular. The disease has no effect on the life of the animal. The subject chosen was twenty-six years old. It is probable that the disability increases with age, and it is certain that in less severely affected animals it is compatible with a useful life. The muscular spasms in stringhalt are most pronounced in the hind limbs. In forward movement there is little interference with normal gait; but in backing and rotation pronounced spasms of the hind limbs occur. The spasms are of two types: (a) flexor spasm, in which the hind legs are flexed and abducted; (b) extensor spasm, in which the hind limb is fixed in extension. Attempts at backing are often impossible to the badly affected animal, owing to the occurrence of alternate flexor and extensor spasms. Similarly, rotation

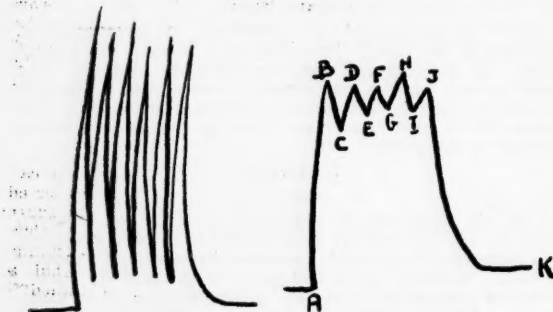


FIGURE III.  
Myotonic gripping before immersion.

FIGURE IV.  
Myotonic gripping after immersion.

is hampered owing to the spasm of the hind legs. The normal horse progresses in rotation by abduction and adduction of the hind limbs round the fixed forelimbs. In the case of the animal affected with stringhalt, the front limbs, being less severely affected, are moved round the immobile spasm-ridden back legs.

It was not possible to demonstrate improvement on repetition of the backing and rotation, but it was observed that after the horse had been led for two miles he could be backed for twenty yards without the occurrence of spasm, and, furthermore, that rotation was much freer. In addition it was noted that the condition was aggravated by fright or excitement, and that it was worse in cold weather or after a change from hot to cold weather.

With the help of Mr. P. Schinckel, B.V.Sc., who examined the subject before and after each experiment, we investigated the response of the animal to drugs.

### Method of Experiment.

Days of approximately equal temperature were chosen. The animal was backed vigorously and the number of steps taken before spasm became evident was noted. Efficiency in rotation was similarly measured. The drug was then administered and improvement or otherwise was assessed on the number of steps which could be performed before spasm occurred.

Dosage was assessed by Mr. Schinckel on the empirical basis of ten times the maximum human dose. All drugs were administered by venepuncture. The drugs administered included the following: "Syntropan" (dose 10 millilitres; supplied by the firm of Hoffmann-La Roche); quinine dihydrochloride (dose 7.3 grammes; supplied by the firm of F. H. Faulding and Company); "Prostigmin" (dose five millilitres; supplied by the firm of Hoffmann-La Roche).

All three experiments were repeated and comparable results were obtained. Identical injections were given to a normal control animal of the same type and build.

### Results.

"Syntropan."—Before the injection of "Syntropan" the legs were in spasm immediately on backing. Rotation was clumsy. Fifteen minutes after the intravenous injection of 10 millilitres of "Syntropan" backing was freely performed for 30 steps before spasm occurred, and rotation could be performed efficiently. The improvement was maintained for thirty minutes, and sixty minutes after the injection the condition of the horse was as before. The injection of 10 millilitres of "Syntropan" into a normal horse was without effect.

Quinine Dihydrochloride.—Before the administration of quinine dihydrochloride spasm occurred after five steps and rotation was poor. A dose of 7.3 grammes of quinine dihydrochloride was injected intravenously. Within two minutes the animal became excited and restless. Spasms of the hind legs were present when he was at rest. Tremors affected the whole body, backward movements were attempted, but spasms were so severe that the animal nearly fell. On forward movement gross incoordination manifested itself, and exaggerated stringhalt spasms resulted from his attempts to regain equilibrium. This state persisted for twenty minutes, and sixty minutes after the injection the horse's condition had returned to normal. Quinine dihydrochloride in the same dose was injected into a normal animal. No departure from the normal was observed.

"Prostigmin".—Before the injection of "Prostigmin" the horse's functional condition was bad. Backing produced spasm of the hind legs after two steps. A dose of five millilitres of a solution of "Prostigmin" (Hoffmann-La Roche) containing 2.5 milligrammes of "Prostigmin" per millilitre was injected intravenously.

Four minutes after the injection spontaneous spasms of both hind legs occurred. After six minutes spasm was apparent after the first step. The legs could not be lifted at all and were dragged along the ground. This condition remained unchanged for forty-five minutes and was accompanied by salivation and diarrhoea. On one occasion the spasm was so intense that the animal fell. Rotation was impossible. Recovery was not complete for a further forty minutes. The experiment was repeated after the animal had been walked for two miles, when his functional condition was

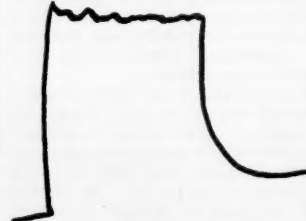


FIGURE VI.  
Myotonic long grip.



FIGURE V.  
Normal gripping before and after immersion.

good; backing without spasm was possible for twenty steps. Within five minutes spontaneous spasm of the hind legs was evident and the symptomatology was similar to that previously described. The effect of the drug lasted eighty minutes. A dose of five millilitres of the same strength "Prostigmin" solution was given to the control animal without effect on the muscular function. Frequent bowel actions indicated that the drug had been administered in quantities sufficient to produce pharmacological action.

#### Comment.

A biopsy was performed on the animal. Examination of the specimen revealed evidence of sarcosporidia infestation, and it was unsuitable for investigation.

It will be seen that not only clinically, but also in its response to drugs, the disease in the horse suffering from stringhalt presented a striking likeness to paramyotonia. Previously we stated that the effect of quinine on paramyotonia was equivocal, and that its administration resulted in signs of idiosyncrasy including mental confusion and vertigo. Its administration certainly resulted in incoordination and vertigo in the horse, and the repeated attempts to balance itself produced additional spasms. The response of the animal to "Prostigmin" conforms to the known worsening effect of that drug on both myotonia and paramyotonia. Finally, we consider that "Syntropan" had comparable effects in paramyotonia and stringhalt. In both the improvement was definite but fleeting.

In our opinion, therefore, paramyotonia and stringhalt are comparable diseases. The effects of drugs in the two conditions are compared in Table II.

TABLE II.

Showing Comparative Findings of Myotonia, Paramyotonia and Stringhalt.

| Clinical or Experimental Finding.               | Myotonia.                  | Paramyotonia.              | Stringhalt.                                    |
|---|----------------------------|----------------------------|--|
| Familial tendency .. ..                         | Yes.                       | Yes.                       | Yes.   |
| Localization to muscles ..                      | Yes.                       | Yes.                       | Yes.   |
| Initiation of spasms on intended movement .. .. | Yes.                       | Yes.                       | Yes.   |
| Good prognosis with regard to life .. ..        | Yes.                       | Yes.                       | Yes.   |
| Predominance in lower limbs ..                  | Yes.                       | Yes.                       | Yes.   |
| Lack of pathological changes in muscle .. ..    | Often.                     | Often.                     | In this case.                                  |
| Worsening effect of cold and fright .. ..       | Yes.                       | Yes.                       | Yes.   |
| Improvement with repetition of movement .. ..   | Yes.                       | Yes, except during attack. | Yes.   |
| Typical electrical reactions ..                 | Yes.                       | Yes.                       | Not tested.                                    |
| Myotonic irritability .. ..                     | Yes.                       | Yes.                       | No.  |
| Reaction to drugs:                              |                            |                            |  |
| (a) Quinine .. ..                               | Condition improves.        | Probably no effect.        | Condition worsens, as shown by incoordination. |
| (b) "Prostigmin" .. ..                          | Condition worsens.         | Condition worsens.         | Condition worsens.                             |
| (c) "Syntropan" .. ..                           | Not tried, lack of stocks. | Condition improves.        | Condition improves.                            |

#### PART III: EXPERIMENTAL INVESTIGATION.

We have endeavoured to show, by analysis of clinical, genetic and pathological data, that the diseases under review are merely variations of one underlying disorder. Studies in goats and horses have given further impetus to that conception. In order to complete the study we have attempted to determine the precise nature of the disorder and its site of action. With this end in view two series of experiments on the paramyotonic subject were decided upon. The first comprised a study of muscle behaviour under certain physiological and physical conditions and in response to the action of drugs, the second an electromyographic recording of the muscle contraction. Similar investigations have been carried out earlier in cases of myotonia and other conditions, and we were thus enabled

to draw upon the experience of various authors.<sup>(43) (45) (48) (79) (80) (82) (86) (87)</sup> No such studies have been made previously in paramyotonia.

The subject, Mrs. Bow., is a member of the paramyotonic family. She deserves the greatest praise for her cooperation and for the keen interest she has shown throughout this sometimes painful and always tedious series of experiments.

#### Myographic Experiments.

We decided first to investigate the effect of cold. Accordingly the right arm was immersed up to the elbow in water for fifteen minutes at 8° C. This manoeuvre led to profound muscular disturbance. It was evident that if we could then measure the rate of recovery after removal of the arm from the bath, it would be possible to estimate the effect of drugs by their influence on this recovery rate.

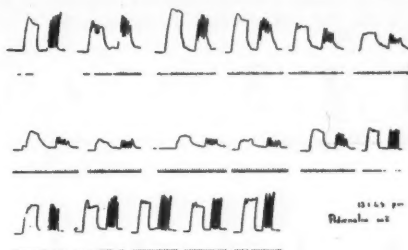


FIGURE VII.  
Myotonic gripping after prolonged immersion at 8° C.

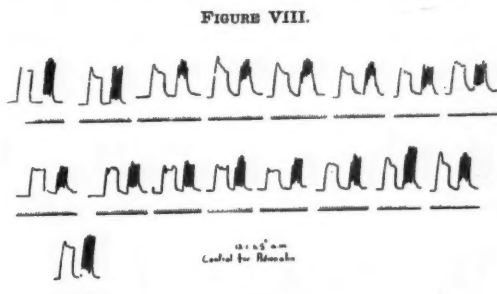


FIGURE VIII.

FIGURE IX.

Myograms from a paramyotonic patient showing (Figure VIII) the adverse effect of adrenaline on the post-immersion recovery period. Figure IX is the record of an identical experiment without adrenaline. The first tracing taken after immersion is the second group in Figure VIII and the third in Figure IX. The first tracings were taken before immersion. Figure IX shows the difference obtained by fifteen minutes' acclimatization to the room, the first tracing having been taken immediately on the subject's entering the room and the second fifteen minutes later. The arm was then immersed for fifteen minutes at 7° C. and the tracings were taken at roughly two-minute intervals thereafter (see text).

We therefore devised a standard exercise which was recorded on a revolving drum. The subject was told to grip a partially inflated rubber bag as tightly as possible for fifteen seconds, and then to relax as completely as possible. This procedure was followed by five grips in succession. The bag, which was inflated to a pressure of five millimetres of mercury, was connected to a rubber tambour working a pointer on a rotating drum. Deflection upwards denoted gripping and deflection downwards denoted relaxation. The deflection was proportional to the strength of the grip. The patient performed this exercise before immersion, after immersion and at fifteen-minute intervals subsequently for periods ranging from two to three hours.

The results indicate that there are wide departures from the normal tracing in the paramyotonic subject both before and after immersion, and that these differences are constant in character and vary only in degree. They are as follows.

1. In the long grip (Figure VI), (a) the plateau of the curve is not well maintained and may be irregular, (b)



the downstroke shows a constant lag in return to normal, characterized by a rounding of the curve at its junction with the base line, (c) the height of the tracing is diminished, which indicates paresis (Figure VI).

2. In the five-grip tracing (Figures III, IV and VII), (a) there are fluctuations in the height of the tracing, (b) the return to the base line is incomplete in one or more tracings, (c) the final grip shows a lag in return to the base line, (d) the duration of the tracing is longer than normal, (e) the height of the tracing is sometimes diminished.

The characteristics of these myograms remained remarkably constant over the 3,000 tracings taken. Clearly two disabilities are present: (i) diminution of the force of grip after immersion, (ii) inability to relax adequately.

In order to measure these features and record them graphically, we made the following measurements, to which we have given the following arbitrary names: (i) average

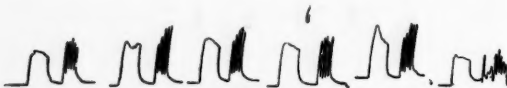
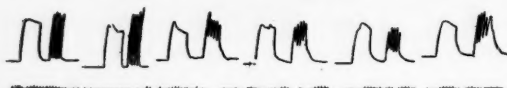


FIGURE X.



Cold control, 19.1-65

FIGURE XI.

Myograms showing the effect of hyoscine on paramyotonia. Figure XI, the control, is essentially the same as Figure X.

excursion, (ii) the work done. Average excursion represents the ability of the subject to move her hand in and out, and is computed thus (Figure IV):

The work done, or the distance the patient moves her hand, is computed thus:  $AB + \text{average excursion} \times 4$ . In comparing tracings such as those in Figures IV and VII it is necessary to have these two measurements. In the latter the excursion may show small departure from the pre-immersion tracing, while the work done would show large differences.

Using these methods, we were able to obtain a graphic record of the rate of change of function in response to temperature.

1. By taking tracings from the arm placed in a series of baths of progressively lowered temperature (allowing suitable intervals between tracings for recovery), we showed that the degree of muscular disturbance was proportional to the temperature, and that this disturbance could be reversed by placing the arm in a hot bath (Figures XVI and XVII).

2. By taking tracings from both arms we found that the effect of temperature was confined to the immersed arm. The right arm was immersed and muscular disturbance was manifest in it, while the unimmersed left arm showed no change. Later the right arm was reimmersed and after

the second bath profound and increasing disturbance was present in it without any effect on the unimmersed arm.

3. The temperature of the room in which these experiments were performed was reasonably constant over the period. It was necessary to allow an interval for acclimatization of half an hour before tracings were taken. In order to preserve as constant an environment as possible, these experiments were repeated in two successive years at the height of summer. All these precautions were necessary because of the subject's extreme sensitivity to changes in temperature.

In investigating the response to drugs, therefore, it was decided to perform the standard experiment in the mornings—namely, to take tracings before immersion after acclimatization to the room, after immersion, and at fifteen minutes intervals subsequently for two hours. The rate of change was measured and recorded graphically. In the afternoon the experiment was repeated and the drug was given immediately after the post-immersion tracing. Subsequent tracings were taken as desired. In experiments in which more frequent tracings were considered necessary, the control experiment consisted of a similar number of tracings.

As an example of a drug which had no effect on the subject clinically or graphically, hyoscine ( $1/100$  grain) is given (vide Figures X, XI, XIV, XV). The results of these experiments are summarized as follows.

**Drugs and Agencies Improving Muscular Efficiency.**—(i) "Syntropan", (ii) heat, (iii) venous congestion, (iv) "Moryl" (at first).

**Drugs and Agencies Reducing Muscular Efficiency.**—(i) Cold, (ii) "Prostigmin", (iii) adrenaline, (iv) strychnine, (v) "Moryl" (after thirty minutes).

**Drugs and Agencies Having Doubtful Effect.**—(i) Potassium citrate, (ii) quinine, (iii) arterial occlusion.

**Drugs Having no Effect.**—(i) Atropine, (ii) ergotamine tartrate, (iii) alcohol (ethyl), (iv) "Benzedrine", (v) phenobarbital, (vi) hyoscine, (vii) benzocaine to the skin.

Certain of these results need further discussion.

#### "Prostigmin."

No record of previous injection of "Prostigmin" in paramyotonia has been made; but observers<sup>(44)(70)</sup> have reported decided worsening effects in myotonia. "Prostigmin" was given twice, the first time in a dose of one millilitre and the second in a dose of 0.5 millilitre. On each occasion serious disturbance arose within fifteen minutes of the injection. The patient's condition simulated a severe attack with cramps in her back. In addition, there was a feeling of tightness in the diaphragmatic region, and difficulty in talking. Her condition grew worse and one millilitre of "Syntropan" was injected, which led to improvement within ten minutes. On the second occasion 0.5 millilitre was given. The disturbance again became alarmingly severe; she had respiratory distress, complete stiffness in her limbs and "wooden" muscles. As before, one millilitre of "Syntropan" was given; its effect was obvious in fifteen minutes and practically complete in sixty minutes.

#### Quinine.

Quinine is known as a dramatic cure for myotonia. Its effect on paramyotonia was unsatisfactory. In this subject symptoms of idiosyncrasy appeared after 0.75 gramme of quinine sulphate had been given, and she presented herself for experiment twelve hours after taking the dose without having taken the second dose of 0.75 gramme prescribed for the morning. The results showed that quinine had no ameliorating effect. If quinine had a comparable effect on paramyotonia to that on myotonia, we believe that some improvement would have occurred. The results obtained here are comparable with the effect of quinine on strynghalt.

#### Adrenaline.

Owing to the rapid destruction of the adrenaline in the tissues and its fleeting physiological effect, it was necessary in this experiment to make observations at frequent intervals. In both the control and actual experiments tracings were taken at intervals of 2, 4, 6, 8, 10, 14, 19, 25, 30, 40, 50, 60, 75, 90, and 105 minutes. A dose of 0.75 millilitre of a 1/1,000 solution of adrenaline (Parke,

Davis and Company) was injected subcutaneously immediately after the post-immersion tracing had been taken. The result was to produce paresis and delay in relaxation within eight minutes of injection, and the effect was pronounced for twenty minutes (Figures VIII, IX, XII, XIII).

# "Syntropan."

"Syntropan" is a product of Hoffmann-La Roche. One millilitre of "Syntropan" contains 10 milligrammes of phosphate salt of the tropic acid ester of a tertiary amino alcohol. We found that it hastened recovery after exposure

effect. The only drugs which seem to influence the condition are those which have in common the feature of some action at the motor end-plate region. These include "Prostigmin", adrenaline and potassium. The precise mode of action of "Syntropan" in this connexion is not understood.

All this, coupled with the finding that the effect of cold is localized to the limb exposed, suggests that the defect lies either at the motor end-plate or in the muscle fibre. In an endeavour to elucidate this point further we decided to perform certain electromyographic experiments.

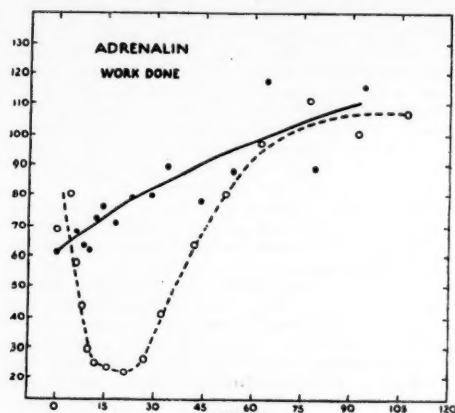


FIGURE XII.

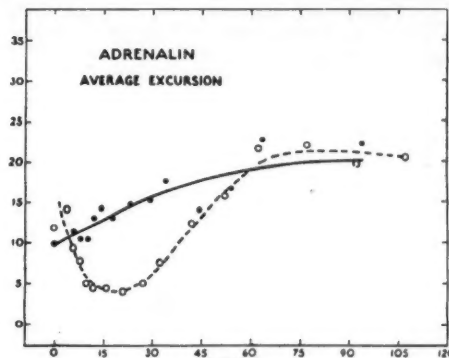


FIGURE XIII.

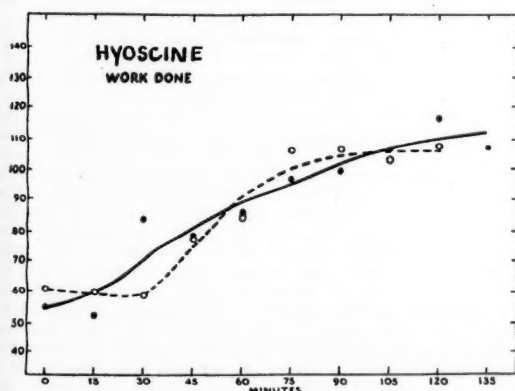


FIGURE XIV.

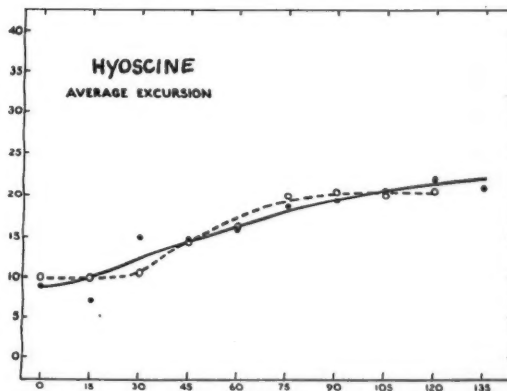


FIGURE XV.

Graphs obtained from measurements of the myograms. The full line represents the control experiment. The adverse effect of adrenaline is clearly demonstrated, whereas it can be seen that hyoscine has no effect on the condition. The two different methods of assessing the myograms give similar results. The ordinates are in arbitrary units (see text).

to cold, and that when given prior to immersion it lessened the disturbance, and functional recovery was more rapid.

# "Moryl."

"Moryl" (Savory and Moore) is a preparation of carbaminyl chlorine, supplied in ampoules containing 0.00025 gramme per millilitre. The action of "Moryl" is that of acetylcholine. "Moryl" led to an improvement, followed by a worsening of the condition, and then to a gradual return of function. The worsening coincided with the advent of sweating and consequent lowering of skin temperature.

# Summary.

To summarize briefly it may be said that no drug primarily influencing the central nervous system, such as "Benzedrine" or phenobarbital, has any effect. No drug which has an inhibitory action on the autonomic nervous system, such as atropine or ergotamine tartrate, has any

# Electromyographic.

The purpose of the electromyographic study in paramyotonia was to obtain corroborative evidence supporting the thesis that the initial defect lay in the muscle fibre. We hoped to rule out the possibility that the condition was due to an upset in reflex innervation or to a pathological change in the central nervous system.

Electromyography consists of the study, by means of suitable electrodes and amplifiers, of the electrical activity associated with muscular contraction. We used concentric needle electrodes inserted into the muscles as suggested by Adrian in 1929. In the literature on the electromyographic study of myotonic conditions there has been some difference of opinion with regard to the meaning of the records obtained.<sup>(1)(2)(3)</sup> We thought that the confusion existing might be clarified by simultaneous tracings from the flexors and extensors of the normal and myotonic grip. The muscles mainly studied were the *flexor sublimis* and

the *extensor communis digitorum*. The location of the electrode was checked by observing the contraction resulting from a stimulus applied to the central wire of the electrode in the selected muscle.

The concentric needle samples the activity of a small portion of the contracting muscle, and this sample may be taken to indicate the activity of the whole muscle. Each motor unit as it contracts has associated with it a change in potential, which is usually referred to as an action potential or action current. This potential change occurs in thousandths of a second and approaches one millivolt in magnitude. As more and more motor units contract,

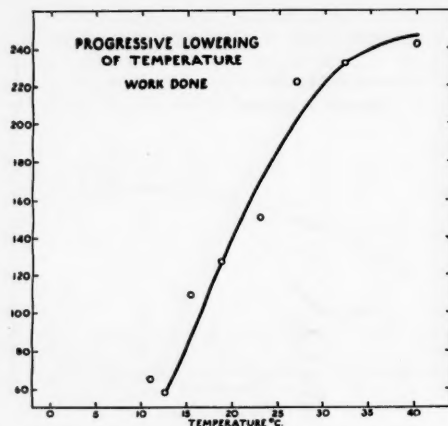


FIGURE XVI.

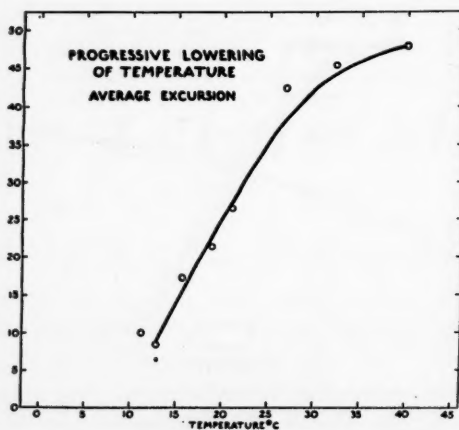


FIGURE XVII.

The effect of a progressive lowering of temperature at fifteen minute intervals by steps of about 5° C. The arm was immersed for fifteen minutes, removed from the bath for fifteen minutes and then reimmersed at a temperature 5° C. lower. Even after the extensive exposure to cold represented at the bottom of the graph, the effects would be almost completely reversed by fifteen minutes' exposure to water at 45° C.

more and more action currents are recorded until even at very slight tensions the record is a mass of spikes. From this stage the record is too complicated for the degree of contraction to be assessed from the number of spikes present.

When an electromyogram is taken, it is customary to place the patient in such a position that the muscle concerned is absolutely at rest. If some action currents are still observed despite the position, either the patient is instructed to relax further or a slight adjustment of

position is made until electrical silence is obtained. The subject is instructed to grip, and the resultant activity is observed. In the case of paramyotonia, the flexor muscles show no signs of activity at rest, but after gripping and relaxing there are signs of activity until the patient is conscious that relaxation is complete. In other words, the slowness of relaxation is associated with muscular activity in the flexors. If the same procedure is carried out in the extensor muscle, one finds that at rest there is no activity, but when the flexion occurs the extensors also contract, as is shown by the action currents. Then when the patient tries to extend the fingers, the action currents increase in density. This can be seen from Figures XVIII and XIX; the instructions given were (from rest) "grip, relax, extend, relax". It can be seen that on gripping and extending movements action currents are set up in both flexor and extensor muscles, and that the duration of the action currents follows the observed duration of the finger movements. It may also be noted that in the relaxed phase between gripping and extension the action currents fall to a minimum; thus the electrical activity is only associated with attempted movement.

Attempts to explain this distribution of muscular activity other than in terms of an upset in reflex activity seemed at first difficult. The position was clarified by a study of the normal relationship.

If single tracings are taken from a normal person, first from the flexors and then from the extensors, a perfect picture of reciprocal innervation can be obtained; but if a simultaneous tracing is taken, we have found (contrary to our expectations) that this is not usually the case (Figure XXI). If a normal grip was executed, a similar distribution of action currents was obtained, as in the paramyotonic patient (Figures XVIII and XIX); that is, there were action currents in the extensors on flexing and in the flexors on extending. A possible explanation can now be offered.

The use of single electrodes and alteration of the patient's position until electrical silence occurs do not guarantee that identical positions of the forearm will be assumed in the study of the two muscle groups. When a simultaneous tracing is taken and the identical position is assured, it becomes apparent that the forearm is a much more complicated piece of muscle machinery than anticipated. It was only by a stilted and awkward movement of the fingers that a tracing approaching that required by the concept of reciprocal innervation could be obtained (Figure XX). In the natural grip the agonist of the fingers may rapidly change to a synergist of the wrist. Thus in the normal and in the paramyotonic patient the same relationship of action currents exists between the two opposing muscle groups in the normal grip. In the latter case the duration is longer than the observed movement. In the forced movement which the paramyotonic patient has to make to extend the fingers, it is obvious to the observer that the wrist synergists are involved.

If the distribution of action currents is normal, this militates against any explanation based on upsets in reflex innervation. Likewise, if the fault was due to an imbalance centrally induced, one would not expect to find a normal distribution of action currents. Another significant observation is the cessation of action currents if the patient relaxes all muscular effort (that is, just stops extending or gripping and rests). It is only when the voluntary effort is being made that the paramyotonia is manifested.

The only difference in the actual spikes recorded from the normal and the paramyotonic patient lies in the overall appearance of the electromyogram. The normal electromyogram is more regular and the spikes are evenly distributed both in height and in density. The paramyotonic record is "ragged", and there seems to be a preponderance of large spikes. These are purely qualitative observations, and unfortunately cannot be considered as indicative of any specific cause. They are merely suggestive of some departure from the smoothness of the merging of many motor units into a sustained contraction.

If it is considered that the paramyotonic muscle fibre is so predisposed—that it will under certain conditions



respond repetitively to the normal nerve stimulus—then in the voluntary effort of gripping this repetitive response may cause the delay in relaxation. It does this because of the distribution of the unusual excess activity in both flexors and extensors and delay in the initiation of the next activity—namely, extension.

To summarize, the following statements may be made.

1. Electromyographic studies by means of simultaneous records from flexor and extensor muscles of the forearm suggest that in normal people it is not usual to obtain dissociated activity in the supposed antagonist. Because of the complex integration of the forearm, it appears that the prime mover of the fingers may become a little later a synergist of the wrist. This finding is contrary to the usual ideas concerning the function of these muscles.

2. The results of electromyographic studies on a paramyotonic subject differ in no obvious way from the normal with regard to the distribution of action current activity in the various groups of muscles during a movement. When the subject was asked to extend the limb after

Contracture has been defined as a state of contraction in muscle which persists for an abnormally long time after the stimulus for its production has been removed.

Tetanus, on the other hand, is a persistent state of muscular contraction which is the result of, and dependent upon, stimulation of a motor nerve above a certain frequency. Contracture has been produced in the forearm of man by strong electrical stimulation.<sup>(71)</sup>

Various types of contracture have been described: (i) veratrine contracture, which occurs in frog's muscle after immersion in veratrine solution, (ii) Bremer's contracture, which occurs in frog's muscle with suitably spaced electrical stimulation; (iii) Tiegel's contracture, which occurs in frog's and man's muscle after extremely strong electrical stimulation.

It has been suggested<sup>(6)</sup> that a contracture may set up the same contractile mechanism as an ordinary contraction, and it has been shown<sup>(21)</sup> that the metabolism is the same. Therefore, contracture would seem to differ from contraction only in the nature and duration of the intermediate processes constituting protoplasmic excitation. Moreover,

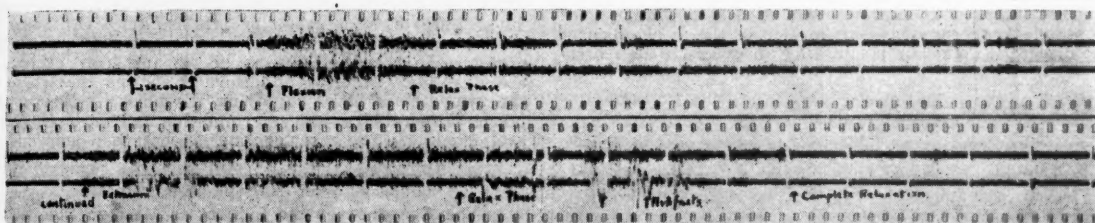


FIGURE XVIII.

Electromyogram from a paramyotonic patient with severe disability. The gaps in the tracings are at intervals of one second. In all the electromyograms the film speed is the same. The top tracing represents the activity in the extensor muscles and the bottom tracing that of the flexors.

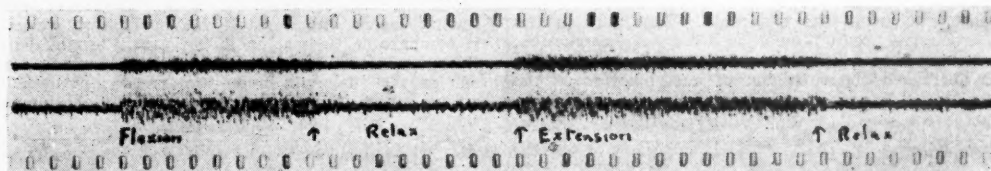


FIGURE XIX.

Electromyogram from a paramyotonic patient with a minimum of disability. The distribution of action potentials is the same as in Figure XVIII, but the duration differs. The action currents can be seen to be a minimum in both tracings in the relax phase just before extension is attempted. The electrical activity is associated only with attempted movement.

gripping, instead of the action currents ceasing quickly as in the normal person, they were prolonged. Simultaneously the patient stated that the muscles were not relaxed. The cessation of the action currents' activity coincided with the time at which the subject felt and stated that relaxation had occurred.

3. The action current activity follows the state of contraction of the muscles concerned. When the subject is mildly affected the movements are performed efficiently and quickly, and the action current activity corresponds. When the subject is severely affected the movements are performed slowly and without much power, and once again the action current activity corresponds.

4. The general impression of the electromyogram when the subject is severely affected suggests that the action current activity differs from the normal, inasmuch as there is a preponderance of larger spikes with a decrease in general density rendering the general picture "ragged".

#### DISCUSSION.

For many years the similarity has been noted between the myotonic contraction and the contraction of normal frog's muscle in response to the drug veratrine. Bremer and Mage<sup>(6)(7)(8)</sup> were the first to study the phenomenon of contracture and apply their knowledge to myotonia.

the tendency to contracture may be associated with ontogenetic and phylogenetic antiquity of muscle, which has been determined by a study of contracture tendency in various frog and animal muscles.<sup>(37)(38)(39)</sup> On this basis, aptitude for contracture would therefore characterize a primitive evolutionary state of muscle. Such a state is not present in normal human muscle.<sup>(6)</sup> However, in myotonic myopathies and in Wallerian degeneration it is suggested that the electrical responses revert to a more embryonic type.<sup>(40)</sup> Furthermore, the myograms of newborn animals are similar in type to myograms of the myotonic patient.<sup>(40)</sup>

If contracture is examined further, it appears<sup>(40)</sup> that there are other similarities to myotonia—for example, the following: (i) contractures decrease on repetition as does myotonia; (ii) quinine decreases myotonic, veratrine and neuro-muscular contracture; (iii) "Prostigmin" increases myotonic, veratrine and neuro-muscular contracture. Therefore there is some evidence to suggest a similarity between the myotonic contraction and contracture. Arguing on these lines, we submit that myotonia may well be regarded as the result of an evolutionary state of muscle in which the response to stimuli is of the nature of a contracture. Before this theory is adopted, it is necessary to consider work<sup>(40)(70)</sup> which has sought to place the defect

at the neuro-muscular junction. From this viewpoint it is interesting to note that in our patients certain muscles, notably those of the eye and the abdomen, have never been affected by myotonic disabilities, even during parturition. It is difficult to imagine a defect in the neuro-muscular

TABLE III.  
(After Ravin.)

| Type of Contracture. | Decreases after Repetition. | Improves with Quinine. | Atropine.  | "Prostigmin." |
|----------------------|-----------------------------|------------------------|------------|---------------|
| Myotonic ..          | Yes.                        | Yes.                   | No effect. | Increases.    |
| Tiegel's frog ..     | Yes.                        | Yes.                   | Decreases. | Increases.    |
| Tiegel's man ..      | Yes.                        | —                      | —          | —             |
| Under veratrine ..   | Yes.                        | —                      | —          | —             |
| Neuro-muscular ..    | Yes.                        | Yes.                   | Decreases. | Increases.    |
| Paramyotonic ..      | No.                         | No.                    | No effect. | Increases.    |

mechanism being present in some muscles and absent from others.

If the theory of humoral transmission of nerve impulses to muscle is accepted, an explanation of the mechanism of the disease is found by postulating an excessive concentra-

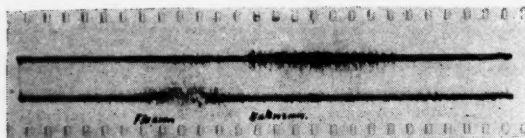


FIGURE XX.

Attempt by a control subject to reproduce a pure flexion-extension movement of the fingers. A very stilted finger movement is needed.

tion of acetylcholine at the end plate. This may be the result of excessive production or of decreased destruction. Similarly, *myasthenia gravis* may be explained as the result of decreased concentration of acetylcholine at the end plate.

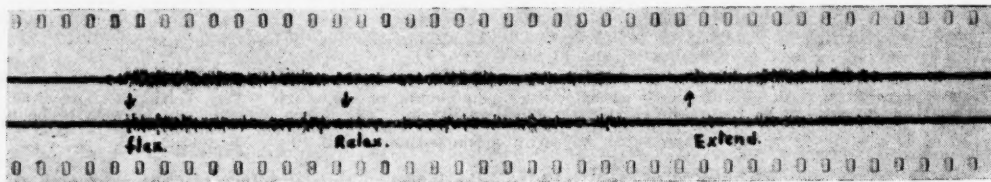


FIGURE XXI.

The electromyogram obtained from a control subject when the fingers are flexed and extended in the usual manner. The distribution is similar to those of the paramyotonic patient (Figures XVIII and XIX). The amplification of Figure XXI is much less than that of Figures XVIII and XIX.

It has been proved that the myotonic contraction can occur when the muscle is divorced from the central nervous system,<sup>(40)(41)</sup> and that transfusion into a myotonic patient of compatible myasthenic blood has no effect on the abnormal contraction.<sup>(45)</sup>

Superficially these facts appear to support the purely chemical theory of production of the abnormal muscle contraction; but there are serious objections to this theory.

The work of Brown and Harvey<sup>(42)</sup> and of Harvey<sup>(43)</sup> suggests that the myotonic muscle is defective in its response to normal quantities of transmitter, and that it, in fact, responds repetitively to any stimulus whether this is mechanical, electrical or chemical. Taken in conjunction with Bremer's conclusions on the ontogenetic relationship of contracture tendency in muscles, such a belief would be an adequate explanation of the fundamental defect in the myotonic contracture. It would lead to the conclusion that the stimulation of muscles in myotonia

and paramyotonia resulted in a repetitive response of the contracture type.

In support of this conclusion observations on the experimental work done on the paramyotonic subject may be considered.

#### Experiments on Response to Cold.

The results from experiments conducted on Mrs. Bow, in regard to the response of her muscles to cold may be summarized as follows. (a) Lowering of the general environmental temperature has an adverse effect on the contraction. (b) The muscular disturbance is proportional to the temperature of the bath in which the arm is immersed. The disability resulting from cold is reversible by heat. (c) The immersion of both arms leads to severe myotonic disturbance and paresis.

Menstruation and cold produce vasoconstriction in the peripheral circulation.<sup>(46)</sup> Both make the subject worse. Adrenaline and fright both have the effect of constricting the peripheral vessels. They also cause vasodilatation of the vessels in the muscle substance. It has been proved that they exaggerate the muscle disturbance in paramyotonia. Stimulation of the vasodilator nerves of a denervated limb or of a group of muscles in a cat leads to contracture,<sup>(42)(47)</sup> and it has been suggested<sup>(48)</sup> that the presence of vasodilatation is accompanied by excess of acetylcholine. Bülbbring and Burn<sup>(49)</sup> showed that some of the sympathetic fibres supplying the blood vessels in the muscles liberated at their termination not adrenaline but acetylcholine. The acetylcholine present then acts on the abnormal muscle, which responds in a contracture-like manner to the stimulus.

#### The Response of the Subject to Various Drugs.

##### "Moryl."

The administration of "Moryl" leads to initial improvement followed by decreasing efficiency. It is known that the earliest effect of "Moryl" is to produce peripheral vasodilatation.<sup>(50)</sup> We would regard this dilatation as resulting in an entirely opposite set of circumstances to that following vasoconstriction. In fact, peripheral vasodilatation is comparable to heating of the arm. It may be assumed that the known action of "Moryl" in increasing muscular efficiency<sup>(51)</sup> depends upon its parasympathetic-mimetic action, which produces an excess of acetylcholine at the end

plate. This state of affairs, while favourable to normal persons, is anything but favourable to the subject with myotonia and results in contracture of the muscle fibre.

##### Adrenaline.

The administration of adrenaline produces an aggravation of muscular disturbance in Mrs. Bow, even leading to paralysis. Part of the known action of adrenaline is to produce vasoconstriction in the skin and vasodilatation in the muscles. Adrenaline increases the efficiency of normal muscle possibly by enhancing the activity of acetylcholine already present or by promoting more efficient production. Consequently the presence of adrenaline results in an increase of acetylcholine at the myo-neural junction. The relative excess acting on the sensitive muscle fibre results in what has been termed "acetylcholine paralysis".<sup>(52)(53)(54)</sup>

In this experiment the effect of the adrenaline was fleeting; but when environmental cold or fright is the

stimulus for adrenaline production, the secretions are continuous, leading in these cases to the progressive worsening of the condition and the ultimate weakness complained of by these patients.

#### Quinine.

The results from administration of quinine are equivocal. As quinine has such dramatic effects in myotonia, it is considered that should its action be pharmacologically comparable in paramyotonia, then even the small amount of drug administered should have produced some improvement.

An explanation for this failure may be given. It has been repeatedly suggested that myotonia is a less severe manifestation of paramyotonia, and that both are due to the underlying abnormal muscle fibre. In myotonia the contracture tendency is less than in paramyotonia. The action of quinine in alleviating the symptom myotonia is thought to be as follows: (i) quinine increases the ability of the muscle to hold a tetanic spasm; (ii) quinine decreases the excitability of the end plate; (iii) the repetitive response of normal muscle to eserine is removed.

It is likely that the last-mentioned is the most important factor, and it is likely that if the repetitive tendency was great, quinine might not be effective.

#### "Prostigmin" and "Syntropan".

"Prostigmin" worsens myotonia and paramyotonia. Its capacity to relieve *myasthenia gravis* is due to its ability to cause muscle to respond repetitively to normal amounts of acetylcholine produced as the result of nerve stimulation. It is therefore understandable that the effect of small doses in paramyotonia is to produce the most dramatic worsening. We consider that this selective dramatic action on the muscles points to an abnormal muscle fibre as the underlying cause of the myotonic disturbance. "Syntropan" has been shown to increase the rate of recovery of the muscle spasm induced by cold. It has also been shown to be rapidly effective in relieving the muscular symptoms produced by "Prostigmin". No work has been done which gives any more accurate indication of its pharmacological site of action; but this result points to an action on the neuro-muscular junction.

#### Electromyographic Experiments.

The study of the electromyograms of the normal and paramyotonic subjects (as produced by double beam cathode ray oscilloscope taking simultaneous tracings from flexor and extensor muscles in each instance) reveals no great difference between the one and the other.

In the normal person it is not usual to obtain completely dissociated activity of one muscle group, and in the paramyotonic subject the same phenomenon occurs. Therefore it is unlikely that any fundamental upheaval in the accepted concept of reciprocal innervation is acting in this disease. When the subject is badly affected the qualitative characteristics of the action currents are altered; this supports the contention that in paramyotonia the defect is of the muscle fibre and has no connexion with the central nervous system.

#### SUMMARY.

1. A clinical study has been made of two families, one suffering from paramyotonia and the other from *myotonia congenita*.
2. A clarification of the clinical picture of the various myotonic disorders has been attempted.
3. A genetic survey of the affected population has been made and discussed.
4. An investigation has been made into an hereditary disease of horses, stringhalt, with which a strong resemblance to paramyotonia is suggested.
5. Experimental investigations on paramyotonic subjects have been made as follows: (a) a pharmacological survey, (b) electromyographic studies.

#### CONCLUSIONS.

1. The myotonic group of diseases include *myotonia congenita*, *myotonia atrophica* and paramyotonia. They are the result of an abnormal muscle fibre of a primitive

evolutionary type. The abnormality consists of a tendency to respond repetitively to stimuli. The tendency is greatest in paramyotonia and least in *myotonia atrophica*.

2. The only drugs which have any profound effect on these diseases are those which have some action upon the myo-neural junction—namely, "Prostigmin", adrenaline and quinine. It is assumed that "Syntropan" acts similarly.

3. The distribution of action currents in relation to muscular movement is the same in normal and in paramyotonic subjects. There are qualitative differences between the action currents obtained from the two subjects, and their presence lends support to the theory of the abnormal muscle fibre.

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### CRIMINAL RESPONSIBILITY.<sup>1</sup>

By W. E. AUDLEY,

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UNDER the processes of the law all adult persons are considered sane and are fully responsible for the offences they commit until it can be shown that they are less than sane or not fully responsible.

Medical opinion is that there is no clear-cut dividing line between sanity and insanity. Between those who are within the range of normality and those who are certifiably insane there is another group who are not normal persons yet are not certifiably insane. This group includes mental defectives, persons who have some degree of dementia, be they young or old, and persons who are in the early stages of a developing mental disorder, and some others. All accused persons of this intermediate group should have their mental capacities estimated at some time before sentence is passed, and in my opinion this time is before the accused is asked to plead. For this purpose all such accused persons, with the exception of those dealt with summarily, should for some weeks prior to the trial be under psychiatric examination by a court psychiatrist, who should render a report to the trial judge or be called to give expert evidence at the beginning of the trial.

In some cases alleged offenders are not committed for trial but are remanded for medical observation. I consider that these persons, some of whom can be certified as insane, should not be certified insane and sent while unconvicted to a criminal mental hospital; but I believe that whenever possible such persons should go to trial unless they are so obviously insane that a jury would reject them at once. For, as will be shown later, "fitness to plead is something less than sound mind", and many such persons are fit to plead. When bail can be granted, one condition of the bail might be that the accused should spend it under observation in a psychiatric hospital or psychiatric annexe to a hospital. This was done recently in the case of a woman who murdered her two children and attempted to commit suicide. Under treatment in hospital she became sane enough to be fit for trial.

Figures given by authorities vary as to the proportions of offenders who are normal, mentally defective, insane and in the intermediate group. Norwood East recently stated that mental defectives numbered 1 in 200, insane persons 1 in 100, and the others who were not normal almost 1 in 5. In view of this, the issue of fitness to plead should be raised before every trial for a major offence, and evidence should be given by a psychiatrist as to the present mental state of the accused.

A person might be considered fit to plead if he fulfilled the following criteria: (i) he was not acutely manic or acutely depressed; (ii) he could make a rational application for a trial; (iii) his behaviour was not so irrational that a layman from his own observations would consider him insane. However, this is a medical opinion, and fitness to plead is a legal opinion, as was stated in a recent judgement as follows:

Whether a person is fit to stand his trial is a legal question, as is the question whether he is responsible

for his action, a question on which his state of health is necessary but not decisive. In my judgement a man may be held fit to plead even though he could not be held to be of sound mind.

If the above criteria for fitness to plead were accepted, there would be very few persons in criminal mental hospitals who had not had a trial and were there merely on remand. These persons would be those who were found unfit to plead. But it would not be necessary to have them detained in a criminal mental hospital. As they were unconvicted and had had no opportunity to defend themselves against the *prima facie* charge made by the police, they should be in an ordinary mental hospital, where their prospects of recovery might be enhanced, and they might later be found fit to plead.

When the question of fitness to plead is disposed of, the accused should come to trial. At the trial the question whether or not the accused person did in fact commit the alleged offence should be decided and the verdict of the jury should be either guilty or not guilty. It should not be competent for the defence counsel to raise the issue of insanity during the trial. After the verdict, but before the sentence, if the accused was found guilty, defence counsel might then bring evidence to prove insanity at the time of the offence and this would then be determined and an order made as required. The order would be to gaol, if the accused was sane then and now, or to a criminal mental hospital if he was insane then and now, whilst if he was sane now but insane at the time of the offence he would be sent to gaol, awaiting the Governor's pleasure.

A person whose sanity was in doubt and who was found guilty, especially if the crime was murder, should be given a sentence at the discretion of the judge, in the light of the medical evidence put forward as to the person's mental state. A person found not guilty would be released, and if at any later time he was found to be insane he would be committed to an ordinary mental hospital under the non-criminal sections of the *Lunacy Act*.

The foregoing discussion stipulates certain minimum requirements for fitness to plead, in the hope that as many persons as possible may have the benefit of a trial. An approach from the opposite point of view to that of fitness to plead was made by Norwood East in 1936, in his book "Medical Aspects of Crime". He then considered a prisoner unfit to plead if he was unable to understand the following six criteria: (a) the nature of the charge against him, (b) the difference between a plea of guilty and one of not guilty, (c) the evidence of witnesses, (d) the procedure of the court, (e) how to instruct his legal advisers, (f) how to take an intelligent part in the trial.

These criteria demand much more of the accused than what has been put forward here, and any psychiatrist who considered that his patient complied with those six points would no doubt also consider him of sound mind. Thus a number of persons who were insane but fit to plead (as mentioned in the judgement quoted) would be denied a trial and would have to be detained without it till a considerable mental improvement was made. However, in 1944 Norwood East, in an address to the London Medical Legal Society, seemed to have recast his ideas, for he stated that if a medical formula of criminal responsibility was introduced it might result in hardship to offenders and embarrassment to psychiatrists. He believes that psychiatry is now sufficiently advanced to be able to support the principle of modified culpability and that what is needed is not a reform in the law but an improvement in our evidence as forensic psychiatrists.

I believe that the three requirements set out by me—namely, that the accused is not elated or depressed, that he can make a rational application for trial, and that his conduct is not obviously irrational—will permit a greater number of alleged offenders to be tried. I believe that the questions "Was the person guilty of the offence?" and "What was his mental state at the time?" should be dealt with separately. It is important for the individual and the community that his guilt be determined. His subsequent disposal and future treatment are even more

<sup>1</sup> Read at a meeting of the New South Wales Branch of the British Medical Association on May 29, 1947.

important to the community and are of especial concern to a society such as is gathered here.

The new Swiss Criminal Code of December 21, 1937, makes the provisions shown in the appendix, and is well worthy of consideration.

#### Appendix.

**Sec. 2. Responsibility. Art. 10. Lack of Responsibility.** Whoever because of insanity, idiocy, or grave disturbance of mental competency at the time of commitment of the offence shall be incapable of recognizing the illegality of his act, or whoever by these reasons shall be incapable of acting in accordance with his insight into the legality of his act, shall not be punished.

**Art. 11. Partial Responsibility.** If the offender, at the time of his act, was mentally disturbed or his mental competency diminished or if he was mentally retarded to the extent that his capacity to recognize the illegality of his act or his capacity to act in accordance with this insight was diminished, the court, in its discretion, may impose a less severe penalty (Art. 66).

**Art. 12. Exception.** The provisions of Art. 10 and 11 shall not be applicable if the serious disturbance or retardation of the mental competency of the offender was occasioned by himself with the intent of committing the offence while in that mental condition.

**Art. 13. Mental Examination.** The prosecutor or the court, when in doubt as to the responsibility of the accused, shall order a mental examination of him by one or more experts. A mental examination shall be ordered if the accused is a deaf-mute or if it is alleged that he is an epileptic. The experts shall state the condition of the accused and also give their opinion whether he requires care in a mental hospital and whether his condition is dangerous to public security and order.

**Art. 14. Detention of Incompetents, etc.** If the incompetent or partially responsible offender is considered so dangerous to public security and order as to necessitate his confinement in a mental hospital, the court shall order his detention. The court shall suspend the execution of the sentence against a convicted person who is partially responsible.

**Art. 15. Care of Incompetents.** If the condition of the incompetent or partially responsible offender requires his treatment and care in a mental hospital, the court shall order that treatment and care. The court shall suspend the execution of the sentence against the convicted person who is partially responsible.

#### CRIMINAL RESPONSIBILITY.<sup>1</sup>

By E. P. DARR,  
Katoomba.

THE subject upon which I was asked to speak was "criminal responsibility", which I have taken to mean who, or what class, or what condition in our society is responsible for producing our criminals. If criminals are born rather than made, if they are predestined to crime as the fundamentalist believes that the heathen is predestined to hell, then we can disclaim all responsibility, beyond the long-range one of beginning the search for the hereditary causes of this predestination. But as all modern criminologists are satisfied that the criminal is made rather than born, we must search for the causes before we can fix the responsibility.

We find that the great majority of our adult criminals have begun their careers in childhood or youth; Mr. Oswald Barnett, of Melbourne, made a six years investigation into juvenile delinquency, and established a number of very important facts. On a population basis, five out of every six young delinquents sent from Melbourne to the Castlemaine prison reformatory were from the inner suburbs, where poverty and overcrowding were at their worst; 68% were unemployed, 17% were in blind-alley jobs, 95% had left school before they were fifteen years old. One might be tempted to expect at this stage that their intelligence was less than that of the young criminals

from the outer suburbs and the country; but the opposite is the truth, for investigation showed that of those from the inner suburbs 31.6% were below normal intelligence, from the outer suburbs 46.2%, and from the country 65%. The only possible conclusion seems to me that the harsher the environment, the greater the proportion of intelligent young people which is driven to crime.

Mr. Barnett found that the Castlemaine reformatory was excellently run, with the aim of educating, fitting with a trade and reclaiming the young delinquent; yet after six years 62% of those from the inner suburbs had been recommitted, and 42% of those from the outer suburbs—these figures again suggesting that the harsher environment was responsible. But long before Mr. Barnett's investigation there was strong evidence that environment—especially poverty—was one of the main causes of crime, for an analysis in the early part of the nineteenth century demonstrated that the amount of theft varied directly, and almost exactly, with the rise and fall in the price of wheat.

From poverty, poor education, dead-end jobs and unemployment must come feelings of frustration, especially for those with intelligence and initiative which they have not the scope to use. Any psychologist knows that frustration gives rise to aggression, and in the circumstances which we have been considering aggression will often show itself in the "basher" gang, robbery with violence and violent sex crimes. In considering the causes of these crimes, Henry Rhodes makes the following statement:

Every youth leaving school emerges with at least a spark of initiative, of individuality, of vision. He then encounters exactly the same conditions as any of his fellows, the machine that does not require his skill, the blind-alley occupation making no demands upon his initiative, and for the edification of his leisure, the cinema with its cheap, mass-produced thrills. That is all that the most advanced civilization the world has ever seen has to offer him. That is how and why the young criminal is produced. And there are respectable people and apparently otherwise humane people who think to cure him with forty strokes of the birch.

I believe that we must recognize in the cinema one of the contributing causes of the increase in sexual crimes; a recent analysis of Hollywood films showed that about 90% had sex as their main theme. Of course, we all know that sex must play an important part in all normal lives; but it should not have that overwhelming importance which Hollywood gives it; young people who constantly absorb these utterly false values are likely to grow up with an unhealthy attitude to sex, especially if they are without the protection of a sane education.

However, I think that this type of crime comes from two main groups of persons. The first has a psychological twist, whether inherited or acquired. We know that the fetishist, the masochist and the sadist are all capable, in the right circumstances, of committing sex crimes; we know that the young man who has an unaccountable and overwhelming impulse to steal a woman's handkerchief may end by committing rape and murder, if he is given the wrong treatment. Rhodes, speaking of such a sexual offender, says:

Corporal punishment had sharpened sadistic instincts; his mental life in prison was a long drawn-out sexual fantasy. Such men emerge from the operations performed by an outraged justice a thousand times more diseased and dangerous than they were before.

No one with the slightest knowledge or experience of the matter can any longer doubt that conventional penal methods are useless against such anti-social acts as these.

The second type of criminal, and probably the much larger class, is recruited from the youths who have been badly educated in regard to sex. If they come from slums their knowledge will be precocious and evil, because overcrowding has made a decent sexual reticence impossible. If they come from the middle class, prudish parents will have left them uninstructed. Again to quote Rhodes: "Too little sexual reticence . . . and too much have a like effect, and inevitably so, since the result is a defective

<sup>1</sup> Read at a meeting of the New South Wales Branch of the British Medical Association on May 29, 1947.



education in one of the most vital matters affecting our lives."

Another part of the problem to be considered is how we treat the criminal. We have possibly outgrown the idea of punishment as revenge, but we certainly have not abandoned the idea of punishment as a deterrent; yet anyone who has given the most cursory study to the history of crime and punishment must know that even the most brutal punishment has never been a deterrent; the inevitable deduction from the story of crime in the eighteenth and early nineteenth centuries is that brutal punishments increase brutal crimes. I am sure that one reason why severe punishments do not act as a deterrent is that men do not stop to think: "What will the punishment be if I am caught?" The criminal expects to "get away with it", and a large number of crimes are committed on the impulse of the moment, without any thought.

We all know that we reclaim only a minority of our criminals, and the reason is that the idea of punishment as a deterrent still dominates our treatment of them. Any intelligent person who looks into one of our gaols for an hour and asks a few pertinent questions will realize that every reform under such conditions is a miracle. According to regulations the prisoner spends about fourteen hours a day in solitude, in a hideous, bare, badly lit cell; meals are insufficient in everything but quantity; recreation is infrequent and always passive; breach of prison discipline probably means at least a week in solitary confinement, with nothing to do but read a Bible in small print by a dim light, and nothing to eat but sixteen ounces of bread per day. We shall continue to discharge our criminals from prison more anti-social than they were when convicted until we learn that the treatment of the criminal should have two objects only—to make him into a decent citizen, and when this is impossible, to protect society. At the moment it is necessary to urge that flogging will do neither of these things; the youth with sadistic tendencies will not be made into a decent citizen by the lash, but his sadism will be intensified by feelings of revenge, so that when he is released some member of society is likely to pay dearly for society's blunder.

A short outline of the history of three criminals will throw further light on our question of responsibility.

One of the most astonishing forgeries of all time was carried out by Marang van Ysselveere. One morning in December, 1924, he called on Sir William Waterlow, the head of the firm that did the official printing for the Portuguese Government; he presented credentials from the highest State authorities. He gave instructions for the printing of 150,000 notes of 1,000 escudos each, and of 300,000 notes of 500 escudos; for reasons of State the issue was to be strictly secret. As there seemed to be no doubt about his credentials and he was able to give guarantees of gold security, he got his notes and took them back to Portugal, where he opened a bank, in Lisbon, using 200,000 escudos from his forged notes to do it, to have an easy way of circulating his notes. The bank also did legitimate business for a year, when the fraud was discovered because of irregularities which had nothing to do with the forged notes. Waterlow and Sons had to pay £810,000 damages to the Portuguese Government; but the ingenious Marang escaped.

Our second example, Ivar Kruger, built his vast financial structure on paper; as he said: "Isn't it wonderful what you can do with a scrap of paper?" When his edifice was beginning to totter he propped it up with more paper—£28,500,000 sterling worth of forged Italian treasury bills. To get the right signatures for his forgeries he wrote to the Italian Minister of Finance and to the Director-General of State Monopolies; these were the wrong signatures, yet his directors and the bank accepted the bonds.

Kruger explained that the negotiations with the Italian Government had been of so confidential a kind that the fact that Kruger, Toll and Co. held these bonds must not be disclosed in any circumstances.

In the rise and fall of Ivar Kruger we see reproduced the development and decay of capitalist society. He started life as an engineer . . . he was concerned with genuine productive acts; he built in ferro-concrete, devised new methods of construction. And then he began to build his huge edifice of paper. The essence of his crime was that a great part of the financial structure he built up bore no relation to production.

The society in which he lived . . . has followed the same course, and is in danger of being involved in the same ruin. Control has passed from those who worked with hand and brain into the hands of those who realize . . . what wonderful things can be done with scraps of paper.

Manfred Georg ends his study of Kruger thus:

Without the spirit of modern capitalism Kruger would not have been possible. His memory will for good or evil remain knit together with this conception as a warning and an example for a new generation in a new epoch.

These two criminals, to whom might be added Hatry and Stavisky, suggest that the shape of society itself is responsible for the possibility of these gigantic frauds. Our third will illustrate the inefficiency of the present penal code in its most important aspect—the protection of society.

In 1902 Albert Moss, at the age of seventeen years, was convicted of forgery at Dubbo and imprisoned for a year. In 1905 and again in 1906 he was sent to gaol for three months for stealing. In 1911 he stole, was taken to the Reception House, certified insane, and detained for fifteen days. In 1914 he was in the Mental Hospital, Gladesville, for fifteen days, and in the Parramatta Mental Hospital for about a month. In May, 1917, he was in the Mental Hospital, Callan Park, after stealing. In May, 1917, he was in the Reception House and the Mental Hospital, Callan Park, for four months—"quite stupid and helpless". In May, 1918, he was in prison for stealing. In August, 1918, he was in the Mental Hospital, Callan Park, after stealing. He escaped. From December, 1918, he was in the Reception House and in the Mental Hospital, Gladesville, till May, 1920. From May, 1921, he was in the Parramatta Mental Hospital till November, 1922. In May, 1923, he was in prison for stealing. In March, 1924, he was in the Reception House for two days. In September, 1924, he was arrested for trying to induce two little girls into a cemetery and sent to the Reception House and the Mental Hospital, Gladesville; he escaped in January, 1925. In September, 1925, he was in the Reception House for four days. In December, 1925, he was in the Reception House for four days. In January, 1926, he assaulted a twelve-year-old girl with intent to commit rape, and was sent to the Reception House and to Parramatta Mental Hospital; he escaped in January, 1933. In March, 1934, for stealing, he was sentenced to thirty days in prison. In May, 1934, for stealing, he was sentenced to twenty days in prison. In November, 1934, after a robbery, he was sent to the Reception House and to Gladesville; he escaped in March, 1937. In September, 1937, for stealing, he was sent to prison for three months. In January, 1938, for stealing, he was sent to Orange Mental Hospital till September, 1938. Between December 13, 1938, and January 21, 1939, he murdered three men. After these three murders he was pronounced "a moral degenerate of an extreme degree".

The salient points of this record are the following:

(i) that he was sixteen times in mental institutions, escaping three times; (ii) that his attempt to induce the two little girls into the cemetery was a warning, in a man of his mentality, that he was becoming a menace to society—this was over thirteen years before the final tragedy; (iii) that the attempted rape, twelve years before the final act, was a clear statement that he had become a menace to society, and that unless he could be cured he should be permanently controlled. Yet after that he was in and out of prison or mental hospital five times. There is no doubt of where the criminal responsibility lies in this case—primarily with society in general, which is responsible for appointing our law-makers, secondarily with the law-makers, and lastly with those responsible for the efficient working of our mental hospitals.

I think we are now justified in coming to the general conclusion that the community as a whole is responsible for the greater proportion of the crime that plagues it. Even in its present form the community could, if it wished, enormously lessen crime if it would abolish slums, improve education, see that all young people have opportunity for healthy sport and cultural enjoyment, keep unemployment down to a minimum, and in the treatment of criminals make use of all the scientific knowledge that is now available. If we did all these things, which are possible even in the present form of society, it is certain that our record of crime would be greatly improved. But an acquisitive society can never free itself from the kind

of crime which springs inevitably from the over-development of the acquisitive sense; we can only hope for freedom from this—especially the Kruger and Hatry type of crime—when we are willing to pay the necessary price, by making cooperation the basis of society.

Finally, our profession must carry a heavy share of the criminal responsibility, for it knows all these things, but for the most part remains silent.

### Reports of Cases.

#### SYMMETRICAL BILATERAL THINNING OF PORTIONS OF THE PARIETAL BONES.

By J. B. CLELAND,  
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Adelaide.

In October, 1937, an old woman, O.P., aged seventy-one years, died at the Adelaide Hospital in an extremely wasted condition, and her death was attributed to the combined effects of bronchitis and bronchiectasis with probably some cardiac failure and perhaps neglect. Much emphysema of the lungs was present, with some fibrosis at the base of the left lung and some bronchopneumonia at the base of the right lung. Some renal fibrosis, two faceted gallstones and some inflammatory infiltration of the rectal mucosa were present. The right eye was blind, the pupil being closed and displaced outwards.

In the parietal bone on each side a symmetrical depressed area was found, somewhat triangular in outline, with the base of the triangle posteriorly. The depression on the right side measured six centimetres laterally and six centimetres antero-posteriorly. On the left side the lateral



FIGURE I.  
Sketch of side view of patient.

extent was seven centimetres and the antero-posterior extent six centimetres. The inner table was exposed at the bottom of these depressions and was very thin, quite translucent and of the thickness of parchment. There was no bulging or depression on the inner aspect of the skull. It seemed as if absorption of the outer table and diploë of the bone had occurred, leaving a sloping edge measuring one and a half to two centimetres or even more in places before the parchment-like portion was reached. At the post-mortem examination the bone presented a reddish appearance round the periphery. It also seemed as though the ridge of bone in the middle line between the two depressions, an area three centimetres across, was slightly more nodular than normal. The depressions approached within about two centimetres of the sutures with the frontal bone, and they were separated from the parieto-occipital sutures by about three or four centimetres.

In a section of the sloping edge of the affected area were seen a few marrow spaces with blood-forming cells, but no osteoclasts or osteoblasts.

The clinical history of the subject, who had been under the care of Dr. (now Sir) Trent de Crespigny, was admirably taken by Dr. John Ray, to whom I am indebted for the sketches. The woman was admitted to hospital on October 1, 1937, and died on November 17. It was impossible at first to obtain any history from her, and her daughter was mentally unbalanced and the son not very bright. She was lying in bed in a semi-comatose condition. General pigmentation of a bronze tint was present, and a few pigmented patches were seen beneath the *frænum lingue*; the skin was dry and inelastic, so that Addison's disease was considered. The left eye had been damaged and its pupil was eccentric. A swelling present in the left iliac fossa suggested a carcinoma. Incontinence of urine



FIGURE II.  
Sketch of front view of patient, showing the  
ridge-like appearance in the mid-line.

developed, and later numerous moist sounds were noted at the bases of both lungs. Dr. Ray's notes state that the patient had "a most peculiar shape to her head; the posterior parts of both parietal bones are flat and come to the superior suture at a distinct angle. Examine rest of family for like change". (There is no record of the result, if such an examination was made.)

The X-ray report by Dr. Nott was as follows:

Posterior parts of parietal bones are very flat and meet together in midline at a distinct angle. ? Bony change present or any foramina in skull. . . . There is an abnormal appearance on the upper posterior region of the cranium. I am not sure whether this is due to deficient development of the parietal bones or shrinking of the brain itself.

This is a well-known but rare condition. Jonathan Hutchinson<sup>(1)</sup> gives excellent illustrations of it. In the text of these articles he gives an account of the condition under the headings "Symmetrical Depressions in the

Parietal Bones (the So-called Senile Atrophy of the Skull)" and "Depressions in the Parietal Bones in Connection with Syphilis". These plates with the same numbering appeared again in his "Smaller Atlas of Clinical Illustrations".<sup>(2)</sup> F. S. Eve<sup>(3)</sup> describes the condition in an ancient Egyptian under the heading "Symmetrical Atrophy of the Skull", and gives an illustration. David M. Greig<sup>(4)</sup> discusses the condition under the title "On Symmetrical Thinness of the Parietal Bones", and gives beautiful illustrations in colour of affected skulls. He writes as follows:

In the centre of the affected parietal area the diploë has never developed, and this part of the parietal has remained in the same condition as the lower parts of the inferior occipital fossæ where the bone is "thin, semi-transparent and destitute of diploë". It is . . . an abiotrophy. . . It is a defect of growth, "congenital" to that extent, but not present in infancy, because at that period the diploë is non-existent; present in adolescents, but obscured by the thickness of the scalp and by the presence of hair and becoming evident in late adult life when the scalp is relatively thinner and the hair is lost.

Greig gives a long list of references to the condition. A. J. E. Cave<sup>(5)</sup> describes the condition as "Bilateral Thin-



FIGURE III.

Photograph of the thinned parietal bones.

ning of the Parietal Bones". In this paper Cave supports the view that it is a growth defect or dysplasia of the parietal diploë resulting in approximation of the inner and outer tables and a translucency of the bone, consequent upon the thinness of the affected region. He states that it may affect other bones of the skull, and that though it is congenital it does not manifest itself in infancy, because then normally the diploë has not yet appeared. He refers to the various views held, such as that the condition is a senile atrophy, an excessive physiological atrophy or a pressure atrophy, or due to stretching of the occipitofrontalis tendon or to syphilis. In the discussion Professor G. Elliot Smith disagreed with the view that it was congenital; he said that the outer table was thin, the diploë was exposed and then eroded. He considered that some factor, such as pressure on the outer table, caused its absorption. In one period of Egyptian history the condition appeared to have been common, as seventy cases were found in a single cemetery. The individuals were

of a rich class, who wore full-bottomed wigs like a High Court judge.

Jonathan Hutchinson states that examples of this condition are nearly always to be found in pathological museums. There were four in the Dupuytren Museum in Paris, and when he wrote his papers the Museum of the Royal College of Surgeons had three.

#### Discussion.

I have discussed the present case with Professor Goldby, who is inclined to support Greig's view that it is of developmental origin, chiefly on the remarkable symmetrical nature of the lesions. For myself, I am undecided, chiefly because it is so difficult to conceive of any factor that could operate symmetrically in this situation, and usually in this situation alone, so as to cause the bony absorption. If absorption has occurred, then this case may represent an example in which the process was nearly but not quite completed. At the autopsy, it was noted that the bone presented a reddish appearance round the periphery, and in the dried bone the shelving edge is still more deeply coloured than the normal bone. Again, Greig, in describing his case, states that "the descent of the anterior border from the skull level to the floor of the flattened area is steep but not sudden". In the present case I have described the edge as sloping, measuring 1.5 to 2.0 centimetres or even more. Is it possible that, in this



FIGURE IV.

Photograph of the thinned parietal bones.

case, absorption was still going on and had not yet been arrested, the redness indicating some increased vascularity, the bottom of the sloping edge the zone where activity still continued? Microscopic sections of the bone were of no help in deciding on the nature of the process.

In this case, if the condition is acquired, there is no evidence pointing to an external factor, such as a full-bottomed wig, as being responsible. One would have to postulate some metabolic bone-absorbing factor of a selective nature. That bone-absorbing factors of a selective nature do operate at times is shown in those examples of decalcification of the bodies of the vertebrae, and of these bones alone, in elderly persons, which are well known to radiologists. Moreover, one must remember that bone metabolism is always going on, as shown by the early appearance of radio-active chemical constituents in the bones after their administration.

#### References.

- (1) J. Hutchinson: "Symmetrical Depressions in the Parietal Bones (the So-called Senile Atrophy of the Skull)", and "Depressions in the Parietal Bones in Connection with Syphilis". *Archives of Surgery*, Volume V, 1894, pages 228 and 359, Plates CXIII and CXIV.
- (2) J. Hutchinson: "Symmetrical Hollows on the Surface of the Skull", *A Smaller Atlas of Illustrations of Clinical Surgery*, 1895, Plates CXIII and CXIV.
- (3) F. S. Eve: "Symmetrical Atrophy of the Skull". *Transactions of the Pathological Society of London*, Volume XLI, 1890, page 244.
- (4) D. M. Greig: "On Symmetrical Thinness of the Parietal Bones". *Edinburgh Medical Journal*, New Series, Volume XXXII, 1926, page 645.
- (5) A. J. E. Cave: "Bilateral Thinning of the Parietal Bones". *Journal of Anatomy*, Volume LXI, 1927, page 486.



## Reviews.

### PRE-NATAL LIFE.

JOSEPH BARCROFT graced English physiology for half a century. He was a big man, full of vitality, and from his publications and his conversation one always got a notion of the zest with which he lived and worked. During his research life he published the results of his researches in the journals, but every now and again he gave his colleagues a treat by writing a book in which he integrated the results of years of research and was quite prepared to talk at large upon the subject. We have only to remember such books as "The Architecture of Physiological Function" to realize the debt we owe him.

The present book, "Researches on Pre-Natal Life"<sup>1</sup> is Volume I of research on that subject. It is rather to be feared that Volume II will not be published, for Joseph Barcroft died last year. The opening words of the preface were all too true:

This work partakes very much of the nature of a will—I hope not my last. In the days of bombs it seemed to me only the due of the many who had given me encouragement and support, not least the Rockefeller Foundation, that I should set down in some connected form such information as I had accumulated concerning pre-natal life: then, if the bomb came my way, the information, for what it was worth, would remain.

From this we can get some notion of the courage of the man.

The book sets out much of the information which had been gained on fetal physiology largely in Barcroft's own institution and elsewhere under his inspiration. It covers the physical growth of the fetus and the manner of its nutrition. A great part of it is devoted to the respiration of the fetus. This section is particularly stimulating and informative; it follows along the line of Barcroft's life work. Even in the text the man's personality shows up: he can, for instance, tell us that such and such an idea occurred to him while talking in a train on his way home from a meeting, or that he did another investigation because an eminent colleague had nettled him, and from all of these motives clear, precise scientific work arose.

This book is one which every person interested in the subject should read. It is a book which should be put into the hands of every young man who has a desire for research. The scientific discipline is admirably shown and the adventure of science clearly portrayed. This is well up to the standard of Barcroft's previous publications, and a fitting last work of a great career.

### INSUFFLATION OF THE UTERUS AND TUBES.

"UTEROTUBAL INSUFFLATION", by I. C. Rubin, is a reference masterpiece by the world's foremost authority on this subject.<sup>2</sup> Thirty years of experience in pioneering and perfecting the technique of this test enable the author to speak dogmatically and to be listened to with respect, if not awe. Here is not a light meal to be toyed with for brief intervals by the dilettante of general medicine, but strong meat to be digested slowly by those whose speciality leads them to the frequent usage of tubal insufflation.

The anatomy and physiology of the Fallopian tubes are described with much detail and various types of tubal peristalsis are shown to vary in relation to menstruation. It is interestingly demonstrated that the tonicity of tubal muscle can be augmented by the injection of follicle-stimulating hormones, and this perhaps accounts for the success sometimes attendant upon the administration of these in cases of sterility in which no infertility factors can be found. Fresh evidence is adduced to establish the existence of the much disputed utero-tubal sphincter.

Careful study of a chapter dealing with contraindications will eliminate many untoward happenings, and the fact that "tenderness" in the fornices always indicates inflammation is stressed. Following insufflation a good deal of insistence is laid on shoulder pain and subsequent fluoro-

scopic control. While this pain is probably pathognomonic of tubal patency when present, many gynaecologists would not be in agreement with the statement that it is "always present when the tubes are normal".

One of the criticisms of utero-tubal insufflation as distinct from lipiodol X-ray examination of the tubes is that the latter alone will show the site of obstruction in non-patency. That this is not entirely so is interestingly demonstrated by Rubin, who claims to localize the obstructions by careful evaluation of the pain and areas of hyperaesthesia produced during carbon dioxide insufflation.

For a comparison of kymographic tubal insufflation and intrauterine injection of lipiodol and other iodized oils we must take two factors into consideration—diagnostic safety, diagnostic reliability. Under these headings Rubin makes out a strong case for utero-tubal insufflation.

In regard to the safety of the method, lipiodol more readily carries infection than carbon dioxide and is more liable thus to cause obstruction of a previously patent tube. Perhaps this is its greatest disadvantage combined with the occlusive effect of retained lipiodol in partially open Fallopian tubes, which sets up a foreign body reaction followed by definite tissue alteration and complete obstruction. On the other hand carbon dioxide is rapidly absorbed from a sub-phrenic pneumoperitoneum and has none of the irritant physicochemical effects of non-absorbed lipiodol. Repeated therapeutic insufflation is thus much safer with carbon dioxide. In nearly ten thousand collected cases the author found infection following lipiodol injection once in 230 cases, while with carbon dioxide injection infection occurred only once in 1,705 cases.

In regard to the relative accuracy of the two methods, while the use of too much lipiodol causes pelvic peritoneal irritation and adhesions, the use of too little leads to many mistaken diagnoses because of incomplete filling of the uterine cavity and tubes. The pressure of lipiodol needed is stated to be greater than that of carbon dioxide and is usually controlled only by a muscular squeeze of the hand, so that much greater care and experience are necessary when it is used. While this may to a certain extent be so, the arguments are presented in a rather biased fashion for a scientific textbook. Regurgitation of carbon dioxide at the cervix is often most difficult to prevent or even to detect, and lipiodol filling of the uterine cavity can easily be controlled by a film developed immediately before removal of the cervical cannula. The truth, of course, lies in the fact that both methods have their place in sterility diagnosis and must depend for their adherents upon the experience of the operators.

The therapeutic aspects of utero-tubal insufflation in sterility are discussed in an excellent chapter. Chance or accident is excluded as far as possible by rigid criteria, and a good case is made out for this type of treatment. Insufflation may need to be repeated on many occasions. When salpingostomy has been performed, the author recommends that the first insufflation should take place two weeks later.

This book is well set up and printed, and for the specialist in sterility it is one that must be read. For others who can spare the time it will provide many enjoyable hours.

### PSYCHOANALYSIS.

In "Deep Analysis" Dr. Charles Berg reports a case history along psychoanalytical lines in such a way as to enable the reader to obtain a considerable degree of insight into this method of psychotherapy. In addition, he includes a chapter on theory and a glossary of technical terms used by psychoanalysts.<sup>3</sup>

Dr. Berg has a capacity for clear exposition, and combines with this a style that is as arresting as the subject matter is interesting. He describes the thirty years old patient as "exceptionally endowed physically, mentally and scholastically", except that he was addicted to bizarre "isms". This and other symptoms were largely resolved when his main sexual difficulties were experienced in phantasy by transference towards Dr. Berg. The author states that he made certain modifications in reporting the facts; for instance, by giving the history a recent setting when actually he treated the patient "many years ago".

It is pertinent to add that, as Dr. Berg's description indicates only a comparatively superficial working-through of pregenital phases of this patient's childhood, it is hardly likely that members of, for example, the British Psycho-analytical Society, would label this a deep analysis.

<sup>1</sup> "Deep Analysis: The Clinical Study of an Individual Case", by Charles Berg, M.D. (London), D.P.M.; 1946. London: George Allen and Unwin, Limited. 8½" x 5½", pp. 262. Price: 12s. 6d.

<sup>2</sup> "Researches on Pre-Natal Life", by Sir Joseph Barcroft, Volume I; 1946. Oxford: Blackwell Scientific Publications. 10½" x 7½", pp. 306, with illustrations. Price: 37s. 6d.

<sup>3</sup> "Uterotubal Insufflation: A Clinical Diagnostic Method of Determining the Tubal Factor in Sterility, including Therapeutic Aspects and Comparative Notes on Hysterosalpingography", by I. C. Rubin, M.D., F.A.C.S.; 1947. St. Louis: The C. V. Mosby Company. 6½" x 9½", pp. 454, with many illustrations, some of them coloured.

## The Medical Journal of Australia

SATURDAY, AUGUST 9, 1947.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

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### A CAUTIONARY TALE.

ONE of the "peripatetic correspondents" of *The Lancet* has told what he calls a cautionary tale.<sup>1</sup> It is an old tale, but will bear retelling. Our tale-teller remarks that he was very much on his mettle for three reasons. In the first place, the subject—"she"—was exceptionally charming; in the second place, her disability aroused his warmest sympathy; and thirdly, he represented, however imperfectly, the profession in opposition to unorthodox practice. For some weeks "she", a most successful comedy actress, had suffered from "paroxysmal sternutation". One can readily imagine, with the peripatetic one, that obstinate attacks of sneezing would be a serious inconvenience to the lady's professional activities. Be that as it may, she failed to secure relief from her own doctor and from the specialists to whom she had been referred. Then—in desperation we assume—she consulted a West End chiropractor "who attacked the problem by a somewhat ungentle manipulation of her nose, not the least attractive of her features". This happened on a Friday morning and was followed by twenty-four hours of nasal tranquillity. The unfortunate sufferer was so overcome that she went back "to her benefactor with words of gratitude and admiration and appropriate complimentary observations on orthodox practitioners". But the respite was short lived, for Sunday saw a resumption of the affliction and in an aggravated fashion. Our peripatetic story-teller asked the patient a question which he declares was not in the least malicious—"Did you go back and tell him?" As might be expected, she did not, and the comment is: "Thus are reputations made."

While reputation may not perhaps be the breath of life to a practising medical man or woman, it has certainly for him or her a reinvigorating power and acts as a kind of magnet, drawing people to him. At the same time it is curious that reputation leaves many unmoved. If it was possible to bring William Osler back to life and to set him in the full flower of his renown to practise in a two-man

town in opposition to an unknown but competent young practitioner, the unknown young man would have some patients. He would probably, almost certainly, have a very thin time compared with his illustrious colleague, but he would have something to do. Reputations may be good or bad, and to seek a good reputation is not only justifiable but natural and to be commended. At the same time we do well to remember that reputation may not be durable—Shakespeare spoke of it as a "bubble"—and that a bad reputation is likely to be harder than a good one—"The evil that men do lives after them; the good is oft interred with their bones; . . ." The reputation most valued by practising doctors is that enjoyed by them among their colleagues with whom they come into daily contact. A man's daily associates can soon discover the depth of his knowledge; they know the pitfalls, the difficulties and the deceptions of medical practice; they can judge of the sincerity with which patients are treated and will not be far out in their estimate of the thoroughness of treatment. They are, in a word, so placed that they can discern a man's worth as a practitioner and his character as a man. This kind of reputation is the most lasting that a medical practitioner can acquire. But the reputation in our cautionary tale is a reputation among patients, and this is something quite different. It may be well founded on skilled and faithful work, or it may be built up on the flimsiest foundations or even on error. Sometimes personality alone operates—there is something in the patient's make-up that responds to what in the world of the stage they would call the doctor's business. Perhaps we might think of corresponding personalities in terms of the quaint little shuttlecock-like shapes that used to be drawn years ago to explain amboceptor and complement, antigen and antibodies in Ehrlich's side chain theory. The comely lady in the tale must have been astonished at the way in which the chiropractic gentleman twisted her nose; the question of personality may not have had anything to do with the cure. Our tale-teller remarks that shock or an outpouring of adrenaline might have been responsible for the temporary improvement. The personality of the patient, however, must have impressed the nasal manipulator when she returned to thank him for the relief he had given her. As we are told, he had a perfect right to believe in his efficiency—the patient had come of her own free will to tell him of his success and there could be no better proof. And for at least twenty-four hours he had no doubt been lauded to the skies among a large circle of friends and acquaintances.

The moral of the tale should not need much emphasis. Our tale-teller puts it in the following words: "We may smile indulgently at the gullibility of the public. . . . But it is well to face facts. Should we be any better than our unqualified competitors if we heard only of our successes and were denied the chastening influence of confrontation with our failures?" The point is that many of us do not take enough trouble to determine what measure of success has been achieved in our treatment and whether the gains made are temporary or permanent. In some circumstances, particularly in private practice, it may not always be easy to do this. But the attempt should be made. If we wish to believe in our efficiency (our tale-teller's phrase is a good one), we must establish grounds for our belief. What has become known as "follow up" practice is some-

<sup>1</sup> *The Lancet*, February 1, 1947.

times adopted in hospitals, particularly when some special investigation is being undertaken. It is necessary when new medicaments have come on to the market or new methods of treatment have been introduced to the clinician. Every clinician will agree that new products should receive adequate trial before they are adopted for general use, and there will be wide agreement with the contention that, once they have been so adopted, their exhibition in any given case should be determined only by the proper indications. In spite of this it is remarkable how often and how easily fashions in the use of drugs and other preparations arise. In a recent discussion on an entirely different subject in a journal from overseas it was stated that consulting physicians who saw a patient perhaps only once, had little opportunity to judge the result of the treatment recommended for use by the family doctor. The suggestion was that in these circumstances the consultant made arrangements that would enable him to form conclusions likely to be of help to him in the future. The consultant who did not do this would be foolish in the extreme. This is just as true of every other type of medical practitioner. Patients, of course, are unpredictable and they may create for a doctor a reputation, good or bad, that he does not deserve. But into the vagaries of patients in this regard we need go no further. In proportion as a practitioner "follows-up" his patients and studies the results of his treatment will he qualify for a place among other practitioners with a sound reputation. Over and above all this there is one condition that ought not to be forgotten—the practitioner may apply to himself the words of Saint Paul, that he must not think of himself more highly than he ought to think. In another place Saint Paul wrote words that are appropriate to the close of this discussion.

For if a man think himself to be something, when he is nothing, he deceiveth himself. But let every man prove his own work, and then shall he have rejoicing in himself alone, and not in another.

## Current Comment.

### CORTICAL CONTROL OF MOVEMENT.

DR. F. M. R. WALSH'S recent contributions<sup>1</sup> to the subject of cortical control of movement are well known; now he has published the essence of his views in "The Victor Horsley Memorial Lecture" for 1946.<sup>2</sup> In the first paper cited Walshe did good service in summarizing the findings of previous workers and in disposing of some fallacious, but still widely held, views. As an example, he drew attention to the work of Lassek and his collaborators<sup>3</sup> who have shown that the cortical area bearing the "giant" cells of Betz is far from being the sole source of the pyramidal tract, and further, that the Betz cells themselves contribute but a small proportion of the fibres

in the tract. The other fibres probably arise from other "large" pyramidal cells in area 4, from similar cells in area 6 and the post-central gyrus, and possibly from other regions as well. At all events, the pyramidal tract is certainly not the simple "Betz-cell-to-anterior-horn-cell" connexion that was originally conceived. All this, however, is subsidiary to Walshe's main theme which is concerned rather with interpretation of the results of cortical stimulation and their application to the psycho-physiology of man's motor life. His thesis falls into two related parts: one is an objection to a strictly hard-and-fast localization in the "motor" cortex, the other is an objection to the atomistic conception of cortical representation of individual muscles.

In the matter of a fixed localization Walshe undoubtedly has the major facts on his side. Quite apart from variation from individual to individual, both in animals and in man (see Penfield,<sup>4</sup> for example), there is variation in a single individual from time to time and under different conditions. One feature which Walshe stresses is the phenomenon of facilitation: continued stimulation of a locus lowers the threshold for further responses; and in time responses proper to adjoining areas may be evoked as well. This seems to be the mechanism underlying the spread of Jacksonian seizures. Murphy and Gellhorn<sup>5</sup> have shown that the neighbouring response is not due to spread, because it persists if the primary locus is mechanically isolated and must be an inherent factor in cerebral organization. They, in common with other workers, found considerable overlap of the areas which represent different parts of the body, but there is a limit to the extent of this overlap. They conceive of cortical representation as a series of topical condensations of activity within a generally excitable area; each condensation has its focus of maximum excitability which gradually falls off as the electrode passes across an indefinite boundary towards the next focus. It is notable that the extent of overlap diminishes progressively in an ascending phylogenetic scale. Even in man, however, Penfield and others still find a definite degree of overlap in bodily representation. There is some evidence that the foci of representation and the extent of overlap vary in the same individual under different conditions. This may very well explain the fact that really discrete motor defects are rarely, if ever, found in cortical damage.

Walshe's second point—that movements, not muscles, are represented in the cerebral cortex—is, of course, the original postulate of Hughlings Jackson. It is still commonly accepted in clinical neurology and at first sight seems unassailable, for it is movements that children learn as they grow up, every daily motor activity is so clearly a movement, and it is predominantly movements which are destroyed in damage to the "motor" cortex. Thus, Walshe takes great exception to experimenters, particularly of the American School,<sup>6</sup> who claim discrete cortical representation of individual muscles. Nevertheless, practically every experimenter on the brain has elicited contraction of individual muscles by exercising sufficient care, and this applies equally to Sherrington, whom Walshe admires, and to Hines, whom he does not. We may add that this representation becomes progressively more definite and discrete in an ascending phylogenetic scale, and seems to run parallel with the progressive crystallization of bodily representation already referred to. The problem is: does the modern atomistic conception really conflict with Hughlings Jackson's view?

A chemical usually reacts as a molecular entity, but this does not deny the existence of constituent atoms; and the atoms can be recombined to give many and varied chemical responses. This, it seems, is the condition in motor control. The atoms of movement are the muscles, and the common observation that different movements may employ the same muscles in different sequences indicates

<sup>1</sup> F. M. R. Walshe: "The Giant Cells of Betz, the Motor Cortex and the Pyramidal Tract: A Critical Review", *Brain*, Volume LXV, 1942, page 409; "On the Mode of Representation of Movements in the Motor Cortex, with Special Reference to 'Convulsions Beginning Unilaterally' (Jackson)", *ibidem*, Volume LXVI, 1943, page 104.

<sup>2</sup> "On the Contribution of Clinical Study to the Physiology of the Cerebral Motor Cortex", by F. M. R. Walshe, M.D., D.Sc., F.R.C.P., F.R.S., Hon.D.Sc. (National Univ. Ireland); The Victor Horsley Memorial Lecture Delivered at the National Hospital, Queen Square, 27th November, 1946; 1947. Edinburgh: E. and S. Livingstone, Limited. 7½" x 4½", pp. 32. Price: 1s. 6d.

<sup>3</sup> A. M. Lassek and G. L. Rasmussen: "The Human Pyramidal Tract: A Fiber and Numerical Analysis", *Archives of Neurology and Psychiatry*, Volume XLII, 1939, page 872.

<sup>4</sup> W. Penfield and T. C. Erickson: "Epilepsy and Cerebral Localization", 1941.

<sup>5</sup> J. P. Murphy and E. Gellhorn: "Multiplicity of Representation versus Punctate Localization in the Motor Cortex", *Archives of Neurology and Psychiatry*, Volume LIV, 1945, page 256.

<sup>6</sup> "The Precentral Motor Cortex", edited by P. C. Bucy, 1944.



that muscles can be isolated individually from some movements and recombined in others. Thus it appears that the progressive minuteness of cortical representation seen in the evolutionary scale really is related to the increasingly precise motor control observable in the same series of animals. Certainly, we think primarily in movements; these, however, are the biologically important movements learnt in childhood, and we retain little recollection of the endless trials of muscular combinations they involved before they were mastered. However, the middle-aged man who tries to learn golf or dancing gains some appreciation of the trial and error demanded before the desired result gradually crystallizes out of the motor chaos. The acquisition of the learned movements of our youth provides a pool of relatively simple motor responses which suffice for the daily round, but this does not preclude the possibility of adding to the pool other, and more specific, muscular skills, for this is the essence of most athletic activity. And, as is well known, increasing concentration and practice will achieve impressive results in the direction of minutely precise muscular control. It is true that, with the possible exception of the muscles of the middle ear, contraction of any individual muscle implies relaxation of an antagonist, and the combination may thus be called a "movement"; but this is a mere quibble to which can be opposed the argument that relaxation of the antagonist might be mediated through the spinal cord, not the brain.

The fund of movements acquired during youth meets most of the requirements of the vast majority. They are thought of as "movements" because they were acquired to fulfil intellectual concepts, as Hughlings Jackson emphasized, and as is indicated by the occurrence of apraxias in the absence of paralysis. But other and more complex movements can be acquired, and extension of this process takes control down to ever smaller muscle groups, or even to individual muscles. So the small boy learns to abduct his big or little toe, "wiggle" his ears or crack his interphalangeal joints, the gymnast learns to contract either of his abdominal recti alone, or the upper or lower parts of these muscles, and Johannes Müller learned to click his auditory ossicles. None of these accomplishments is considered other than as a "trick" movement—of no particular biological importance—and only the sophisticated appreciate that they are really exploring the possibilities of cortical control of muscles; all the same, the results which are evoked at the periphery must have a cerebral counterpart, for these are all consciously desired and deliberately cultivated activities.

Thus it would appear that there is no essential conflict between Hughlings Jackson's conception and the view of individual muscular representation in the cerebral cortex. Any disagreement is rather verbal than real. The bulk of mankind thinks in movements, partly because biological necessity demands their acquisition as such and partly because few are aware of the elements of which movements are composed. Also, most are content with what suffices, even if it is relatively crude. At the same time, the possibilities of muscular control are very much greater than the majority realize, and the control can be extended in favourable cases down to individual muscles. This possibility links up closely with the fact that individual representation of muscles can be demonstrated experimentally in the excitable cortex. In other words, the mechanism is available, but the extent to which it is exploited depends upon the wishes and mental capacity of the individual.

#### THE DOSE OF A DRUG.

THE report of a conference on "The Dose of a Drug" held among members of the departments of pharmacology and of medicine of Cornell University Medical College and New York Hospital makes interesting reading. The discussion was opened by Harry Gold, who advocated a more intelligent attitude towards dosage in drug therapy. In his opinion a large proportion of the failures in drug

therapy resulted "not so much from the choice of the wrong drug, but from the use of the correct drug incorrectly". He discussed the use, as a basis for therapy, of the "average dose" in the pharmacological sense, that is, "the dose which exerts a particular effect in 50% of the population". This idea was criticized by other speakers at the conference as likely to cause confusion and will not be amplified here. However, it is agreed that basic dosages should be determined as rationally as possible. Gold then went on to discuss the cumulative and non-cumulative dosage plans. The cumulative plan involves giving a small dose at the beginning and repeating it at such intervals as to build up a concentration in the blood or the tissues adequate to produce the therapeutic effects. Thus a full dose whose safety is doubtful can be built up by harmless smaller doses. To determine the correct interval between doses it is important to know when the peak effect of any dose is reached. If this is not determined and observed it is unjustifiable to criticize the therapeutic efficacy of the drug. The non-cumulative plan is just the reverse. It involves the giving of a single effective dose, but its repetition at such intervals as will prevent raising the concentration in the blood or the body tissues. In both these plans it is important to know "how quickly the drug is absorbed, the time it takes to reach a peak effect, and something about the speed of elimination or duration of action". For the majority of drugs, according to Gold, cumulation is a self-limiting process. The period of cumulation varies with individual drugs; when a certain blood concentration has been achieved or a certain effect produced (for example, a certain heart rate following administration of digitalis) the blood concentration or therapeutic effect becomes constant. Several examples were quoted by Gold of the fallacy of not observing these principles. A small dose of quinidine may be prescribed and administered for weeks without effect on the disorder of cardiac rhythm for which it was prescribed; yet the fact that sufficient cumulation has not occurred in the first four or five days to produce an effect should be sufficient indication that the size or frequency of the dosage requires to be increased. Morphine may be given at intervals of ten or fifteen minutes in urgent situations, a meaningless process, since the peak effect of a dose is reached in thirty to forty-five minutes or even longer. The effects of "Prostigmin" wear off in about two hours; it is absurd to repeat at intervals of six hours a dose which produced no effect when first given. The mercurial diuretics are excreted within twenty-four hours; their exhibition at four-day intervals, as often recommended, leaves long gaps in the patient's treatment. "Mapharsen" is almost completely excreted in two to three days; a plan of weekly injections seems illogical. There are other circumstances, Gold considers, in which a fixed plan of dosage is indicated "with the highest prospects of therapeutic benefits and lowest liability of producing toxic effects", this fixed plan being adhered to in all cases without attempting individual adjustments; an example of this is the use of digitalis in the treatment of the heart failure of rheumatic carditis, in which the therapeutic results are often indecisive and attempts at adjustment of dosage are unsatisfactory.

There was a good deal of discussion of Gold's ideas, some of it adversely critical. However, he seems to have demonstrated his main point clearly enough: "If physicians were to make more systematic use of the basic principles of dosage plans, the efficacy of drug therapy would greatly increase." The modern tendency is towards the abandonment of empiricism in favour of the use of drugs whose pharmacological and therapeutic action is understood. It would be difficult to justify the discarding of an empirical drug of widely accepted therapeutic worth and the use of such drugs will probably continue to be dependent on "rule of thumb" methods; but a knowledge of the essential pharmacological properties of the important drugs, where these are known, will repay any practitioner who is prepared to apply the knowledge intelligently. It is a matter for further thought just how far prescribing according to a pharmacopoeia with standard prescriptions and fixed dosages is compatible with the rational adjustment of dosages as advocated by Gold.

<sup>1</sup> *American Journal of Medicine*, March, 1947.

## Abstracts from Medical Literature.

### GYNÆCOLOGY.

#### Primary Sarcoma of the Fallopian Tube.

LEWIS C. SCHEFFEY, WARREN R. LANG AND FRED B. NUGENT (*American Journal of Obstetrics and Gynecology*, December, 1946) report a patient with primary sarcoma of the uterine tube and have summarized the 21 cases of this condition recorded in the literature. The patient was seventy years of age. Curettage had been performed and radium inserted into the uterus in June, 1943, for a sudden severe vaginal hæmorrhage. At this time there was no macroscopic or microscopic evidence of malignancy. Following this the patient continued to have an intermittent blood-stained vaginal discharge. At operation in March, 1944 a small atrophic uterus was found displaced anteriorly by a fixed soft mass in the pouch of Douglas; the mass appeared to come from the left adnexa. Before, and at operation, the diagnosis was thought to be a pyosalpinx and no thought of malignancy was entertained. Complete hysterectomy and bilateral salpingo-oophorectomy were performed and subsequent biopsy revealed a myosarcoma of the left tube; the tumour was of the mixed cell variety and showed considerable anaplastic activity. An analysis of the reported cases of sarcoma of the uterine tube shows an age incidence which coincides with that of the fundal carcinoma and tubal carcinoma group. Vaginal bleeding or a blood-stained watery discharge is the most outstanding sign and pelvic or abdominal pain the most important symptom. On pelvic examination a mass of variable size is usually found. In no instance was the diagnosis of tubal malignant disease entertained before operation. The diagnosis usually made was: ovarian carcinoma, pelvic inflammatory disease, ovarian cyst or uterine fibroids.

#### Carcinoma of the Body of the Uterus.

NORMAN F. MILLER AND CHARLES W. HENDERSON (*American Journal of Obstetrics and Gynecology*, December, 1946), in a study of 322 patients with carcinoma of the uterine body, evaluate accepted methods of treatment and state their results and the treatment which they prefer. For nearly half a century hysterectomy was accepted as the best treatment for carcinoma of the uterine body, but follow-up studies gave no cause for optimism. From 1925 it was found that radiation treatment in advanced cases prolonged life and cured some patients. The literature attests the effectiveness of irradiation in the control of uterine carcinoma. Used alone, neither radium nor X-ray treatment appears to effect results equal to those obtained by surgery, but when irradiation is combined with surgery, especially as a pre-operative measure, the prospects appear very promising. The average age of patients in this series was fifty-four years, and the time lapse from the onset of symptoms to the beginning of treatment was twelve and a half months. Clinical and histological grading was carried out in the case of 301 patients and is of importance in the estimation of the survival

rate. Ninety-six patients were treated according to a set plan of pre-operative deep X-ray therapy followed in six weeks by adequate surgery. Of these patients, 84.7% survived for three years, 77% for five years and 65% for ten years. The authors are of the opinion that pre-operative X-ray treatment is a valuable adjunct to total hysterectomy and bilateral salpingo-oophorectomy in the treatment of carcinoma of the body of the uterus. Its use clears uterine infection, reduces the size of the uterus, facilitates operation and does not interfere with wound healing.

HOWARD C. TAYLOR, JUNIOR, AND WALTER F. BECKER (*Surgery, Gynecology and Obstetrics*, February, 1947) discuss the end results in 531 cases of carcinoma corporis uteri with particular reference to the clinical extent of the disease, the histological type of carcinoma and the type of treatment given. The average age of patients was 57.4 years, the five-year period of greatest incidence was 60 to 64 years, and 73.8% of patients had passed the menopause. It would appear that women destined to develop cancer of the uterine body marry with about the normal frequency, but then show a relatively high degree of sterility. Twenty-two patients had primary carcinoma elsewhere than in the body of the uterus—eleven having carcinoma of the breast and three having carcinoma of the cervix. Abnormal vaginal bleeding, usually slight, was the outstanding symptom and was present in 96.6% of patients. The average duration of symptoms before the commencement of treatment was fifteen months, but there was no evidence that failure of treatment was directly related to the duration of symptoms. The absolute "five year cure rate" for the entire series was 38%, but adjustments for patients who died of unrelated disease and for those of whom track was lost gives an apparent "five year cure rate" of 45%. An analysis of the relationship of clinical extent of the disease to end results is of significance. The "five year cure rate" among group 1 patients (uterus not enlarged) was 66.6%; among group 2A patients (uterus not larger than a ten-weeks gestation) 54%, among group 2B patients (uterus larger than a ten-weeks gestation) 37.9%, among group 3A patients (extension of carcinoma to the cervix) 24.3%, and among group 3B (extension of carcinoma beyond the uterus) 11.2%. According to histological classification the "five year cure rates" were as follows: adenocarcinoma, grades 1 and 2, 47.2%; adenocarcinoma, grades 3 and 4, 22.8%; adenoacanthoma, 51.3%. Among the patients on whom hysterectomy was performed the "five year cure rate" was 51.1%. The question of whether the cure rate from hysterectomy was increased when the operation was supplemented by irradiation could not be answered on the basis of the analysis of this series of cases.

#### Leucoplakia Vulvæ.

MORTIMER N. HYAMS AND OSCAR BLOOM (*American Journal of Obstetrics and Gynecology*, February, 1947) discuss the etiology of leucoplakia vulvæ and submit a preliminary report on the investigation and treatment of eighteen patients with this disease. In all cases the diagnosis was verified by histological examination and each patient was subjected to a thorough physical examination and biochemical tests including blood counts, blood chemistry

analysis, vitamin assays, basal metabolic rate estimation, Wassermann reaction, urinalysis, estimation of oestrogen and gonadotrophic hormone content of urine, and examination of the gastric contents for free hydrochloric acid and total acidity. Practically all tests yielded negative results with the outstanding exception of gastric analysis, which revealed the presence of little or no free hydrochloric acid in approximately 60% of patients. This latter observation was originally made by Swift, of Australia, in 1932, and is of ætiological significance. Clinical studies in other fields have demonstrated the relationship existing between achlorhydria and a low plasma level of vitamin A. Premature infants have little or no free hydrochloric acid in the stomach and a concomitant low plasma level of vitamin A. Diseases of adult life, such as carcinoma of the stomach and pernicious anæmia with achlorhydria, are accompanied by a plasma level of vitamin A which is either low or entirely absent. The vitamin A plasma levels were normal in the eighteen patients studied, and the authors consider that the leucoplakia vulvæ was due to a subclinical deficiency of this vitamin or to failure to use the vitamin A present in the plasma. During the past decade leucoplakia has been considered to be due to an æstrin deficiency, but, in the experience of the authors, æstrin administered in adequate doses not only failed to relieve the local condition, but was often followed by aggravated symptoms. The histological changes in leucoplakia—hyperkeratosis, acanthosis, and inflammatory reaction in the dermis—closely resemble the changes in epithelium following the continued absence of vitamin A from the diet. All patients in the series were given 250,000 to 500,000 units of vitamin A daily by mouth, supplemented by injections of 50,000 units twice weekly. Each patient received fifteen minims of dilute hydrochloric acid in water thrice daily with meals. Improvement was noted by clinical appearances and checked by histological examination after treatment. Of the eighteen patients treated, fourteen were free from signs and symptoms and the other four had associated systemic disease. Biopsies taken after therapy showed thinning of the epidermis, reduction of keratin and a decided improvement in histological features. The authors are of the opinion that leucoplakia vulvæ is not a neoplastic and precancerous disease, but is of metabolic origin and is due to a failure of utilization of vitamin A. Uncomplicated early leucoplakia can be relieved with adequate doses of vitamin A and dilute hydrochloric acid by mouth.

#### Surgical Treatment of the Uterine Fibroid Tumour.

JULIAN WALDO ROSS (*American Journal of Obstetrics and Gynecology*, February, 1947) reports 1,500 patients with uterine fibroids treated surgically, 900 of these having had myomectomy. The uterine fibroid is a benign neoplasm arising as a local hyperplasia in the fibro-muscular component of the uterus, and it should be treated conservatively unless definite indications, such as hæmorrhage, pain, pressure symptoms, rapid growth, signs of degeneration, or interference with pregnancy, call for surgical intervention. Abdominal hysterectomy has been the operation of choice since the first celiotomies for



fibroids were performed in 1843. Myomectomy has fallen into disrepute on account of recurrence of fibroids and following deaths after operation due to hemorrhage, sepsis, sapremia and adhesions. The author states that all patients with uterine fibroids are submitted by him to diagnostic curettage, and when hyperplasia of the endometrium is found endocrine therapy is given. He stresses the importance of careful pre-operative and post-operative treatment and gives the details of the operation of myomectomy for submucous fibroids. It is of importance to recognize the fact that the endometrium is infected in all patients with submucous fibroids. Sulphonamides and penicillin should be used before, during and after operation; chronic infection of the cervix or other foci should be eradicated before operation; the vagina should be prepared as for Caesarean section, and post-operative drainage of the uterus must be ensured by preliminary dilatation of the cervix. Myomectomy is technically more difficult and time-consuming than supravaginal hysterectomy, but the author considers that the preservation of the genital functions with the associated nervous stability and tranquillity outweighs by far any technical difficulties. The author hopes that myomectomy, as described, will be recognized as the treatment of choice for uterine fibroids.

#### Infectious Granulomatous Lesions of the Cervix.

W. F. GUERRIERO, R. JENNETT AND W. B. MANTOOTH (*The Journal of the American Medical Association*, March 22, 1947) include the lesions of tuberculosis, *granuloma inguinale*, syphilis, and chancroid of the cervix as the infectious granulomata. Their importance depends on the resemblance of their microscopic appearances and symptoms to carcinoma. *Tuberculosis cervicis* may be primary or secondary and has an age incidence of twenty to forty years. The lesions may be ulcerative, military, papillary, or bacillary catarrhal. The lesion may extend to the vagina, parametrium, uterus and adnexa. Abnormal bleeding and leucorrhœa are the main symptoms. *Granuloma venereum*, caused by the Donovan body, is relatively common in the Negro. The early lesion is a shallow ulcer and the advanced lesion a large friable cauliflower-like excrescence which may destroy the cervix and extend. The finding of intracytoplasmic Donovan bodies in the monocytes is diagnostic. The symptoms do not vary from those of carcinoma, but the youth of the patient is suggestive of a non-malignant lesion. Sulphonamides applied locally and antimony given parenterally are specific for treatment. Syphilitic lesions are important, since the primary and secondary lesions are often mistaken for erosions and the tertiary for carcinoma. Carcinoma and syphilis often coexist. Dark field examination will diagnose the primary and secondary lesions, and biopsy and serology the tertiary. Cervical chancroid is rare; the cervix is red and oedematous with one or more round, firm, punched-out ulcers containing easily removed whitish exudate. The diagnosis is made by smear, culture, skin test, and biopsy. The symptoms and signs include soreness in the lower part of the abdomen, foul greyish vaginal discharge, and burning and pain in the vagina. Local and parenteral administration of sulphathiazole in the best treatment.

## OBSTETRICS.

### Radiology and Difficult Labour.

J. CHASSAR MOIR (*The Journal of Obstetrics and Gynecology of the British Empire*, February, 1947), in his second Ingleby Lecture, discusses the value of radiography in forecasting the course of labour. The following features indicate a good obstetric pelvis. (i) The obstetric conjugate is adequate in size. (ii) The sacrum shows a full, even curve from above downwards. (iii) The pubic bone and its descending ischial rami are nearly parallel to a line joining the sacral promontory to the sacral tip. (iv) The transverse diameter of the brim is adequate, and the posterior segment of the brim outline is well rounded. (v) The limbs of the pubic arch diverge widely enough to accommodate the head with little wastage of space under the apex of the arch. (vi) The fetal head is moderate in size relative to the pelvis and is favourable in position. The author presents a method of simplifying these criteria by recording the dimensions of the antero-posterior and transverse diameters on charts, one being used for the brim, one for the cavity and one for the outlet. Separate charts were made for each size of the fetal head (as measured by the biparietal diameter) from 9.0 centimetres to 9.9 centimetres; and it was found from a study of a series of deliveries that it was possible to draw up a single set of three charts showing the dividing line for easy and difficult deliveries for each size of the fetal head. The graphs do not cover the following abnormalities: a high inclination of the pelvic brim, flattening of the sacral curve, and malpositions of the head. The author suggests that radiography should be employed in cases in which there is a high head at term with suspected pelvic contraction, abnormal or prolonged labour, post-maturity, prematurity, breech presentation, suspected hydrocephaly, or a history of previous difficult labour; also to allay the fear of obstetric difficulty, and when a *primipara* is to be delivered at home.

### Maternal Obstetric Paralysis.

ACCORDING to John T. Cole (*American Journal of Obstetrics and Gynecology*, September, 1946) the objective and subjective findings in obstetric paralysis are not always constant. Pain during labour, referred along the course of the sciatic nerve and increasing as the uterine contractions become more intense, is the earliest sign. Pain may be associated only with uterine contractions. Various paresthesias, spasmodic contractions and paralysis may occur in the lower limbs. "Foot-drop" is rather a constant puerperal finding, but other muscles of the leg and thigh may be paralysed, and in severe cases atrophy and wasting of muscle groups will follow. The paralysis is due to trauma of the lumbosacral cord by the fetal head or instruments, generally occurs in *primiparae* and is unilateral as a rule. The type of pelvis which predisposes is one in which the posterior ilium is short, the ala of the sacrum has only a shallow anterior concavity, and the promontory does not encroach on the capacity of the posterior segment. The portion of the vertex in relation to the ala compresses the lumbosacral cord when the head seeks an obliquity.

Treatment consists of support of the injured extremity, active and passive motion, galvanic stimulation, vitamin therapy and the use of a walking brace. The prognosis must be guarded, as partial or complete recovery may extend over two or more years.

### Pregnancy with Hypertensive Disease.

LEON C. CHESLEY AND JOHN E. ANNITTO (*American Journal of Obstetrics and Gynecology*, March, 1947) review the pregnancies of 218 patients who suffered from hypertensive disease. The policy of the Margaret Hague Maternity Hospital is not to terminate pregnancy in severe cases, and 301 pregnancies are analysed. The standard for hypertension is taken as 140 millimetres of mercury (systolic) and 90 millimetres (diastolic) or greater. The fetal loss in prior pregnancies was 35%, in first hypertensive pregnancies 38% and in subsequent pregnancies 40%. Fetal loss increased with higher initial blood pressure, second trimester rise in blood pressure, higher pressures near the time of delivery, decreased renal function, proteinuria and superimposed toxæmia. The blood pressure of nearly 40% of the patients dropped in mid-pregnancy; proteinuria occurred in association with half the pregnancies; renal function was normal in 93% and premature separation of the placenta in 5.6%. There were six immediate deaths (2.0%) and seven deaths late in the puerperium. The review establishes the danger of pregnancy in the hypertensive patient—one woman in twenty-three died either immediately or within four months after delivery and two in five lost their babies.

### Deaths from Post-Partum Hemorrhage.

CLAYTON T. BEECHAM (*American Journal of Obstetrics and Gynecology*, March, 1947) states that over a fifteen-year period, from 1931 to 1945 inclusive, the Maternal Welfare Committee of Philadelphia has judged 168 deaths from post-partum hemorrhage which is 8.45% of the total maternal deaths in that period. Of these 121 or 72% were deemed preventable deaths, due either to errors in judgement or technique or by the *accoucheur* or to the patient's carelessness or ignorance. The mortality figures for post-partum hemorrhage have remained constant over this period. Errors picked up by the committee's work were: (i) the attendant's leaving the third stage to an intern or nurse, inexperienced in the signs and symptoms of hemorrhage; (ii) continuation of inhalation anaesthesia through the third stage, although the patient was bleeding excessively; (iii) omission of the palpation or holding of the fundus; (iv) allowing the placenta to remain in *utero* whilst the patient bled to death; (v) neglect of close observation for one hour following delivery of the placenta; (vi) neglect to give energetic treatment before the symptoms of shock appeared. Post-partum hemorrhage deaths were caused by several factors in most cases. Such factors were: carelessness, inadequate pre-natal care, inadequate care during labour and operation, ill-advised operation and meddlesome obstetrics, poor choice and use of anaesthesia, poor management of the third stage of labour, failure to recognize hemorrhage in time, insufficient use of oxygen, of packing and of whole blood.



## Bibliography of Scientific and Industrial Reports.<sup>1</sup>

### THE RESULTS OF WAR-TIME RESEARCH.

During the war a great deal of research was carried out under the auspices of the Allied Governments. It has been decided to release for general use a large proportion of the results of this research, together with information taken from former enemy countries as a form of reparations. With this end in view, the United States Department of Commerce, through its Publication Board, is making a weekly issue of abstracts of reports in the form of a "Bibliography of Scientific and Industrial Reports". This bibliography is now being received in Australia, and relevant extracts are reproduced hereunder.

Copies of the original reports may be obtained in two ways: (a) Microfilm or photostat copies may be purchased from the United States through the Council for Scientific and Industrial Research Information Service. Those desiring to avail themselves of this service should send the Australian equivalent of the net quoted United States price to the Council for Scientific and Industrial Research Information Service, 425, St. Kilda Road, Melbourne, S.C.2, and quote the PB number, author's name, and the subject of the abstract. All other charges will be borne by the Council for Scientific and Industrial Research. (b) The reports referenced with an E number may be obtained in approved cases without cost on application to the Secondary Industries Division of the Ministry of Post-War Reconstruction, Wentworth House, 203, Collins Street, Melbourne, C.1. Copies of these are available for reference in public libraries.

Further information on subjects covered in the reports and kindred subjects may be obtained by approaching the Council for Scientific and Industrial Research Information Service, the Secondary Industries Division of the Ministry of Post-War Reconstruction, or the Munitions Supply Laboratories (Technical Information Section), Maribyrnong, Victoria.

PB 40970. CATTELL, MCKEEN, AND GOLD, HARRY. Pharmacology of BAL. Progress Report, Nos. 1 and 3-5. November, 1942, to October, 1943. 82 pp. Price: Microfilm, \$2.00; Photostat, \$6.00.

These progress reports cover the pharmacology of BAL (1,2-dithioglycerine) and related dithiol compounds for the first year under Contract OEMcmr-245. The observations were made on cats and other animals. The study of BAL also included means for protection against BAL poisoning. The related compounds studied were NDR compounds 139, 132, 230, 397 and 420. Numerous tables and graphs are included. These progress reports cover work performed under the auspices of the Committee on Medical Research of the Office of Scientific Research and Development.

PB 41049. CONN, JEROME W., AND JOHNSTON, MARGARET W. Improvement of ability of soldier to work in humid heat. Monthly Progress Report, Nos. 1-17. November, 1942, to September, 1945. 21 pp. Price: Microfilm, \$1.00; Photostat, \$2.00.

These progress reports deal with laboratory experiments made upon men performing hard work in hot environments (85° F. and 85% humidity). The following investigations were made: the effect of the ingestion of large amounts of water at mealtime upon the appetites of the subjects; function of the sweat glands in the economy of NaCl; salt requirements of acclimatized men; determination of the metabolic mixture (protein, fat and carbohydrate) and its relationship to the salt intake; the negative nitrogen balance associated with the period of acclimatization; and the relationship of salt intake and protein metabolism in fully acclimatized men. Some of the findings are briefly noted. These reports cover work performed under the auspices of the Committee on Medical Research of the Office of Scientific Research and Development (Contract OEMcmr-232).

PB 41032. CRISMON, J. M., AND FIELD, J. The quantitative application of combined low temperature and pulsating external pressure in the prevention of gangrene associated with massive edema in immersion foot, frostbite, burns and other injuries. Monthly Progress Report, Nos. 1-6. September, 1944, to July, 1945. 9 pp. Price: Microfilm, \$1.00; Photostat, \$1.00.

These progress reports deal with the circulatory and other physiological changes associated with frostbite. Studies of the circulation in rabbit ears and hind feet subjected to

severe frostbite were carried out with the aid of fluorescein. The therapeutic effect of closed plaster dressings upon edema or gangrene and the therapeutic effect of pulsating external pressure upon the volume of swollen frostbitten feet were noted. Other studies showed the relationship between the rate of swelling of frostbitten feet of rabbits and the changes in the haemoglobin concentration, haematocrit, arterio-venous oxygen difference, whole blood and plasma specific gravity of arterial blood and venous blood draining from the frostbitten region. Also included are studies carried out upon rabbits' feet to test the effectiveness of the combination of rapid warming together with subsequent application of closed plastic dressings and pressure dressings. These progress reports cover work performed under the auspices of the Committee on Medical Research of the Office of Scientific Research and Development.

PB 41037. DAVIS, HALLOWELL. Effects of noise on hearing. Monthly Progress Report, Nos. 1-7. September, 1942, to July, 1943. 8 pp. Price: Microfilm, \$1.00; Photostat, \$1.00.

Experiments were carried out with human and animal subjects in order to determine the effect of routine exposures to loud tones upon hearing. The experiments were conducted at various frequencies, intensities and duration times. Progress Report No. 7 contains a brief summary of the final report and deals with the hearing-loss, loudness-loss, diplacusis, and articulation-loss. These progress reports cover work performed under the auspices of the Committee on Medical Research of the Office of Scientific Research and Development (Contract OEMcmr-194).

PB 40946. DENNY-BROWN, D., et alii. Factors contributing to disability from civilian head injury (excluding age and alcoholism). Abstract of interim reports. (OSRD CMR Neurosurgery Report 25, 39, 41 and 47. Abstracts.) January to August, 1944. 3 pp. Price: Microfilm, \$1.00; Photostat, \$1.00.

An analysis was made of several hundred cases of civilian head injury with regard to the importance of various factors influencing the nature and duration of consequent disability. All cases were followed from time of admission to the hospital for a period of six months or more. The percentage figures for the various general symptoms are given. These reports are abstracts of final reports and only brief summaries of the findings are given. The reports cover work performed by Fellows of Harvard College at the Neurological Unit, Boston City Hospital, under contract with the Office of Scientific Research and Development (Contract OEMcmr-159).

PB 40948. DENNY-BROWN, D., AND BRENNER, CHARLES. Nerve lesions induced by continued pressure. Progress Report, Nos. 1-7. August, 1942, to July, 1943. 11 pp. Price: Microfilm, \$1.00; Photostat, \$1.00.

A study was made of the motor paralysis and lesions produced by the application of continuous pressure to nerves. The experiments were carried out upon nembutalized cats by compressing a chosen segment of nerve with intact blood supply against a brass plate. Later experiments utilized metal clips which were applied directly to the sciatic nerve (of cat). Various pressures for different periods of time were applied during the course of the tests. The histological changes are noted. These progress reports cover work performed under the auspices of the Committee on Medical Research of the Office of Scientific Research and Development (Contract OEMcmr-160).

PB 47071. DETHIER, V. G., AND CHADWICK, LEIGH E. Rejection thresholds of the blowfly for a series of aliphatic alcohols. 1946. 16 pp. Price: Microfilm, \$1.00; Photostat, \$2.00.

This report describes tests with the blowfly *Phormia regina* Meigen in order to determine the stimulating effect of a series of alcohols after extirpation of the antennae and labella, which are its sole loci of olfactory receptors. The proboscis response after stimulation of the tarsal receptors was noted. All test solutions were prepared with 0.1M sucrose as a base. The results of the tests are summarized in Table 1, together with the values of several properties of the alcohols which are of interest to the interpretation of the data. A high degree of correlation was found between the mean concentrations of the alcohols at rejection thresholds and such properties as boiling point, vapour pressure, molecular surface, molecular moment, water-oil distribution coefficients, standard free energies and activity coefficients. The mechanism of the method of sensory reception is discussed. Bibliography. This report is a contribution from the Medical Division, Edgewood Arsenal, Maryland.

PB 40959. DOWDY, ANDREW H. Clostridium infection in dogs. Progress Report, Nos. 1-18. January 1, 1942, to November 15, 1944; February, 1942, to November, 1944. 22 pp. Price: Microfilm, \$1.00; Photostat, \$2.00.

The effect of drugs upon various clostridium infections was investigated, the therapeutic agents employed being sodium sulphadiazine, sodium sulphathiazole, sulphanilamide,

<sup>1</sup> Supplied by the Information Service of the Council for Scientific and Industrial Research.

penicillin, and Lederle's pentavalent gas gangrene antitoxin. The clostridium organisms were *Clostridium welchii*, *Clostridium novyi*, *Clostridium sordellii*, *Clostridium septicum* and *Clostridium perfringens*. Mixed culture infections also included *Staphylococcus aureus*. Penicillin was seen to be a fine therapeutic agent. Certain experiments showed that there was no incompatibility between sulphadiazine and penicillin. The conditions for the optimum use of penicillin or the pentavalent antitoxin are noted. The comparative immunities conferred by toxoids to the various organisms are discussed briefly. Tables are included. These progress reports cover work performed under the auspices of the Committee on Medical Research of the Office of Scientific Research and Development (Contract OEMcmr-48).

PB 40360. FINK, COLIN G., et alii. Use of metal in internal fixation of human bone fractures. Progress Report, Nos. 1-4, 1-15. February, 1942, to January, 1944. 23 pp. Price: Microfilm, \$1.00; Photostat, \$2.00.

These progress reports summarize the results of tests to determine and evaluate quantitatively the resistance to corrosion by body fluids of metals and alloys employed in bone surgery, and to test the physical and mechanical properties of these metals. The metals tested were tantalum, vitallium, Resistal KA2, Zimmer SMO and Pierce SMO. Attempts to evaluate complicated surface areas by means of a Langmuir film failed. A method for surface evaluation of plating with nickel is described. Mention is made in these reports of progress made in purchasing and developing tools for the experiments. The shape and form of the metals *in vitro* and tests to determine the optimum method of fixation are also considered. These reports cover work performed under the auspices of the Committee on Medical Research of the Office of Scientific Research and Development (Contract M-826).

PB 40364. FULTON, J. F., AND HOFF, E. C. A five-foot shelf on aviation medicine. (National Research Council. Committee on Aviation Medicine Report 43.) April, 1942. 3 pp. Price: Microfilm, \$1.00; Photostat, \$1.00.

This is a bibliography relating to aviation medicine and consists of the following sections: (1) general physiological and medical texts with sound treatment of basic topics such as respiration, oxygen, circulation, aerobismolism and the special senses; (2) monographs and journals on aviation medicine; (3) the more specialized monographs on respiration, exercise, high altitude physiology, and the special senses.

PB 40356. GOREL, WALTHER F. Antigens of Flexner group of dysentery bacilli. Progress Report, Nos. 1-20. September, 1942, to December, 1945. 33 pp. Price: Microfilm, \$1.00; Photostat, \$3.00.

A study was made of several types of Flexner dysentery bacilli regarding the chemical nature and toxicity of the antigens. A comparison was made of the *Shigella dysenteriae* types, V, Z, VZ and the Newcastle and Sonne strains. Methods for the isolation of specific antigenic substances are described. These were identified as toxic lipocarbohydrate-protein complexes. Attempts to obtain the toxic substances free from the protein or carbohydrate constituent failed. The specificity of the antigens, variation in antigen content of different strains, and the stability of the antigens were also studied. The results of mouse and human immunization tests are also discussed. Two graphs show the effect of 0.1N alkali and of ultra-violet irradiation on the precipitability of the type V antigen. Several tables are also included. Some pages will not reproduce well. These progress reports cover work performed under the auspices of the Committee of Medical Research of the Office of Scientific Research and Development (Contract OEMcmr-216).

PB 40494. GREEN, DAVID E. Disinfection of drinking water. Mode of action of chlorinating agents. Bimonthly Progress Report, Nos. 1-3, 5-8. August, 1944, to October, 1945. 16 pp. Price: Microfilm, \$1.00; Photostat, \$2.00.

An investigation was made of the mode of action of chlorinating and other water sterilizing agents. The study covered the biochemical effects of sterilizing agents on the common bacterial contaminants of drinking water and an extension of this study to bacterial spores and protozoan cysts. It was found that chlorine and compounds containing active chlorine irreversibly inactivate enzymes whose activity depends upon the integrity of certain groups which are sensitive to oxidation. These groups are generally regarded to be SH groups. The sterilizing action of chlorine-containing compounds has been shown to be due primarily to the irreversible destruction of the triosephosphoric dehydrogenase which is essential for the oxidation of glucose and hence the growth of the microorganisms. The action of organic mercurials is readily reversed by various reducing agents, whereas that of chlorine is reversed with great difficulty or not at all. The reports also include the preparation of, and tests with, the proteolytic enzyme papain, which is used as a reagent for measuring the concentration of active

chlorine in drinking water. Tables show (1) the effects of various agents upon the oxidation of glucose and viability of *Escherichia coli*, *Proteus vulgaris*, *Chromobacterium prodigiosum*, *Lactis aerogenes* and spores of *Bacillus subtilis*; (2) the effects of exposure time; and (3) the effect of organic mercurials as sporicides and bactericides. These progress reports cover work performed under the auspices of the Committee of Medical Research of the Office of Scientific Research and Development (Contract OEMcmr-443).

PB 47066. HIMWICH, HAROLD E. Medical aspects of fat metabolism. No date. 16 pp. Price: Microfilm, \$1.00; Photostat, \$2.00.

Fats, fatty acids, phospholipids and cholesterol are included in this study. The following topics are discussed: digestion and absorption; fat storage, obesity and emaciation; fatty liver; cirrhosis of the liver; oxidation of fat and ketosis. It was noted that abnormal storage of fat may lead to either obesity or emaciation. Experimentally, conditions resembling cirrhosis of the liver have been produced by one-sided diets containing either an excess of one substance, such as cholesterol, or a deficiency of another, such as methionine. Fatty liver must be regarded as a deficiency disease and treated as such. Ketosis of diabetes is to be combated, although ketosis in *petit mal* epilepsy is used therapeutically. Bibliography. This report is a contribution from the Medical Division, Edgewood Arsenal, Maryland.

PB 47073. PINSCHMIDT, N. W., AND HAAG, H. B. Studies on poisoning with mustard gas: I. Mechanisms concerned with H poisoning in dogs. II. The effects of certain drugs on vomiting and fatalities in dogs given mustard gas by vein. September, 1946. 61 pp. Price: Microfilm, \$2.00; Photostat, \$5.00.

Part 1 of this report is a study which was designed to show that the primary mode of action of mustard gas (H) is probably not in the direction of tissue catabolism; the increased urinary nitrogen elimination after the intravenous administration of mustard gas is probably due to the absorption of blood hemorrhaged into the intestinal tract or to a general moribund condition. In this regard the following mechanisms were investigated: (1) interference with glucose metabolism (glucose tolerance studies); (2) interference with the phosphate energy systems (blood phosphate studies); (3) disarrangement of the acid-base equilibrium (blood gas studies and electrolyte studies); (4) circulatory failure (blood gas studies). The various experiments were performed on the same animal (dog), always using an intravenous LD<sub>50</sub> of mustard gas which was always given at the same level of starvation. Part 2 of the reports describes tests to determine whether certain antiemetic drugs (morphine sulphate, "Sodium Amytal", scopolamine hydrobromide and normal saline solution) might be effective in controlling mustard gas emesis. Tables and numerous graphs are given in the report, together with a bibliography. This report covers work performed under contract with the Medical College of Virginia, Richmond, Virginia.

PB 47065. ROEDER, KENNETH D., et alii. Synaptic conduction to the giant fibres of the cockroach and the action of anticholinesterases. No date. 19 pp. Price: Microfilm, \$1.00; Photostat, \$2.00.

A large number of sensory fibres from the cercus of the cockroach enter the sixth abdominal ganglion and form synaptic connexions with twelve to sixteen giant fibres which ascend the cord. These synapses conduct synchronously up to 400 stimuli per second with a delay of 0.6 to 1.3 millisecond. Spatial summation occurs. Treatment with DFP (di-isofluorophosphate) causes marked synaptic facilitation alternating with periods of synaptic block, a condition which persists after washing in saline solution. The conduction process in the giant fibres appears to be unaffected by DFP in the concentrations used. It is concluded that trans-synaptic conduction is dependent on cholinesterase. The report includes a list of illustrations, which, however, are not included, and a bibliography. The work was performed under contract between the Chemical Warfare Service and Tufts College.

PB 41036. WEST, E. S., AND DAVID, N. A. To study the acute and chronic effects of sulfited foodstuffs and sulfites in animals and man. Bimonthly Progress Report, Nos. 1-2, 4-5. June, 1944, to February, 1945. 4 pp. Price: Microfilm, \$1.00; Photostat, \$1.00.

These progress reports cover experiments on dogs and humans which had been fed sulphited foods over a protracted period of time. The following effects are noted: (1) there is an apparent disturbance of calcium and phosphorus metabolism; (2) there appears to be a slight hypoglycaemic effect of sulphited fruit. Also included is a survey of the literature pertaining to the subject, and a brief description of the necessary laboratory and experimental work. These reports cover work performed under the auspices of the Committee on Medical Research of the Office of Scientific Research and Development (Contract OEMcmr-434).



## Special Article.

### FEDERAL INCOME TAX.<sup>1</sup>

THE financial year having come to a close on June 30, 1947, it now becomes necessary for all members of the medical profession to prepare a return of their income for that period.

No doubt many medical practitioners keep their books on a proper double entry system and prepare an income account periodically. These should find no difficulty in preparing their returns; it is to those who have not done so and who are not well versed in the necessary accountancy knowledge required for the preparation of the return that the following remarks are specially directed.

#### Forms to be Used.

A medical practitioner practising his profession on his own behalf in Australia will prepare a return of his income on a form supplied by the taxation department and described as "Form B—Business and Professional Return".

When two or more persons are practising in partnership, a return will be prepared for the whole of the partnership transactions on a similar form described as "Form B—Business and Professional Return", which should be endorsed "Partnership Return".

The net income shown on the partnership return is not taxable by the department against the partnership; each individual partner is taxable on his share. Details as to the names, place of residence, proportionate share of income to which each partner is entitled and the amount of each share must be given in statement Number 5 on page 3 of the return.

Each individual partner should then prepare a separate return described as "Form A—Personal Return". Each partner must show on this return the amount of his share as set out in statement Number 5 on the partnership return, together with any income received from other sources, except non-taxable income, which includes the following: Pensions (war service, invalid, old age and widows); endowment, under the *Child Endowment Act*; dividends on gold mining shares; ex-Australian income, where such income is taxed outside Australia; deferred pay to ex-servicemen; interest on war savings certificates; lottery prizes; betting gains (unless conducted as part of a business).

A medical practitioner not practising his profession on his own behalf, but in receipt of a salary, will use "Form S" and show the amount of salary opposite item Number 1. If in addition to salary he is in receipt of other income such as interest, dividends, rent *et cetera*, he will use "Form A—Personal Return".

If a taxpayer derives income, other than dividends, from more than one State he will furnish a "Central Office" return which must be lodged with the Deputy Commissioner of Taxation in Melbourne.

#### Income.

**Professional Income.**—Professional income includes the amount of gross income from practice, by way of fees, lodge payments and medicines supplied during the year. This amount will be shown opposite item Number 31 on the return.

If a proper record has been kept of all fees earned during the year this figure will be readily arrived at. If no record has been kept, but all fees *et cetera* have been banked, then the bank pass book will show the amount of gross income for the year.

If all fees *et cetera* have not been banked, then to those actually banked must be added the amount retained and used for expenses, either private or professional.

When fees *et cetera* are used for professional expenses without being passed through the bank account, I would suggest that in future a book be kept for recording such expenses, as it is quite probable that the department may not allow the expenses as a deduction from income unless details are supplied.

**Dispensary.**—If a medical practitioner conducts a dispensary and if the dispensary is run entirely apart from his medical practice, it is, of course, essential that proper records be kept of purchases and sales of medicines *et cetera*, wages and other expenses, if any, in connexion therewith, and that the stock of medicines *et cetera* on hand be listed and priced at June 30 in each year.

<sup>1</sup>This article has been compiled at the request of the Federal Council of the British Medical Association in Australia.

These items of revenue and expenditure in connexion with the dispensary would not be shown on the return as income *et cetera* from the practice, but under the appropriate headings of items Numbers 32 to 36 inclusive.

**Rent Revenue.**—When a medical practitioner pays rent for the premises in which he is carrying on his practice and when any portion of such rented premises is used as a residence, an amount equal to two-thirds of the total rent paid should be treated as income. (Item Number 41.) As the whole amount paid as rent will be claimed as a deduction, the net allowance for rent in connexion with the professional practice is thus equivalent to one-third of the total rent paid.

If it is considered that one-third of the total rent paid is too small a proportion to be allowed in the particular circumstances, the amount which the taxpayer considers fair and reasonable should be stated together with the grounds on which he arrives at such amount.

**Bad Debts Recovered.**—Only such amounts as have been actually written off as bad and claimed as a deduction from income in previous years and which have been recovered during the year covered by the return should be included opposite item Number 37.

Care should be taken by medical practitioners who do not keep a complete set of books, to see that such items are not also included in the gross income referred to above, otherwise double taxation will result.

**Board and Quarters.**—A taxpayer in receipt of a salary who, in addition, is provided by his employer with board and/or quarters, must include as income opposite item Number 2 on the return the value of such board and/or quarters.

**Defence Force Pay and Allowances.**—A medical practitioner who during the year ended June 30, 1947, received any pay or allowances as a member of the defence force in Australia must supply all the details set out opposite item Number 4 on the return.

#### Deductions.

The following items of expenditure are allowable as deductions from income and should be shown alongside the appropriate items numbered 54 to 70 inclusive on page 2 of the return.

**Salaries and Wages (Item Number 54).**—The amount of salaries and wages actually paid to any person in the employ of the taxpayer who is engaged in work necessary for the production of his income is allowed as a deduction. Such persons include an assistant doctor (on a salary), a nurse, a *locum tenens*, a charwoman for cleaning consulting rooms (if rooms are cleaned by a general servant, portion of her wages can be claimed as a deduction under this head), a chauffeur, a groom.

**Food for Employees (Item Number 55).**—A medical practitioner who employs any person exclusively for the purposes of his practice and in addition to the wages or salary of such person paid in money, supplies him or her with food, may claim as a deduction the amount actually expended thereon. If in these circumstances the taxpayer is unable to state the actual amount expended as aforesaid, then such sum is deducted as in the opinion of the Commissioner is just and reasonable.

**Rent (Item Number 56).**—The amount of rent paid for consulting rooms, or if a medical practitioner is practising at his place of residence rented by him, then the total amount of rent paid for such residence, is deductible. (Note reference to this item under heading "Rent Revenue".) The name and address of the lessee of the premises is to be shown on the return.

**Rates and Taxes (Item Number 57).**—If a medical practitioner is renting or leasing property and is using the property as a residence and consulting rooms combined and if the lease provides for the payment by the tenant or lessee of municipal and/or water rates, one-third only of such rates (not including excess meter rates) actually paid during the year can be claimed as a deduction.

If the leased premises are wholly used as consulting rooms the total amount actually paid can be claimed as a deduction.

**Insurance (Item Number 58).**—Any fire, burglary, third party or comprehensive insurance premiums paid for the insurance of any property used in conducting the practice, such as office, consulting and waiting room furniture, surgical instruments and plant, motor-car, horses or vehicles, together with workmen's compensation premiums, may be deducted. A list showing the name and address of each insurer and the amount of premium paid should be attached to the return.

**Interest (Item Number 59).**—The actual amount of interest paid on money borrowed for the purpose of acquiring or



carrying on a practice may be claimed as a deduction under this heading. Name and address of person to whom interest is paid to be shown on return.

**Depreciation (Item Number 60).**—A deduction may be claimed under this heading for the wear and tear of property (other than land and buildings) used for the purpose of carrying on the practice. The rates of depreciation allowed are as follows: (a) bicycles, 10%; (b) buggies and sulkies, 10%; (c) carpets, 10%; (d) diathermy plant, 7.5%; (e) electrocardiograph, 5%; (f) furniture and fittings (consulting rooms), 2.5%; (g) furniture and fittings (hospitals), 5%; (h) high-frequency current machines (surgical), 7.5%; (i) harness, 10%; (j) horses, 10%; (k) library, 5%; (l) motor cycle, 15%; (m) motor-car, 15%; (n) ophthalmic surgeon's plant, 10%; (o) radium plaques and needles, 10%; (p) Röntgen ray machines, 7.5%; (q) typewriters, 10%; (r) X-ray and high-frequency current plant, 7.5%. No depreciation is allowed in respect of: (a) doctors' instruments; (b) horse rugs; or (c) hospital bedding, linen, crockery *et cetera*, but the cost of replacements (not including alterations, additions or improvements) is allowed as a deduction under the heading of "Repairs" (Item Number 61).

Note that depreciation can be claimed only as a deduction at the above rates, calculated on the diminishing value of the assets as at June 30 of the previous year, that is, the cost price of the asset, less the above percentages of depreciation deducted for each year in use in the business.

**Special Depreciation (Item Number 60).**—A special initial depreciation of 20% of the cost will be allowed in respect of plant, machinery *et cetera* acquired or installed during the year ended June 30, 1947, unless the taxpayer elects otherwise. If a taxpayer does not desire this special allowance, he must elect to that effect by notifying the Commissioner in writing on or before the last day for the furnishing of the return of the year of income in which the property is acquired or installed, as the case may be. In addition to the special allowance, depreciation at ordinary rates will be allowed as a deduction calculated on the cost of the property after deducting the amount of the special initial depreciation.

**Repairs (Item Number 61).**—All repairs and replacements (which would not come under the heading alterations, additions or improvements) effected by a medical practitioner at his own cost to any assets employed in carrying on the practice, including covenanted repairs to that part of rented premises used as consulting rooms, are allowed as deductions.

**Bad Debts (Item Number 62).**—If all fees earned have been previously returned as income, irrespective of whether they have been collected or not, and any of such fees still remain uncollected and are irrecoverable and have been actually written off as bad debts, such amount will be allowed as a deduction from the current year's income.

If in the past and also in the present return only the actual fees received have been included as income, then, of course, no deduction can be claimed for bad debts.

**Subscriptions (Item Number 63).**—Amounts paid during the year of income in respect of the taxpayer's membership of a professional association are allowable deductions, provided that the total deduction allowable under this heading shall not exceed £10 10s. to any one association.

**Travelling Expenses (Item Number 64).**—Only travelling expenses incurred in the production of income will be allowed as a deduction; for example: (i) motor-car expenses, cost of running, benzine, oil, tires, garaging *et cetera* (repairs to be shown under separate heading referred to above); (ii) buggy and sulky expenses, horse feed, shoeing *et cetera* (harness and vehicle repairs to be shown under heading "Repairs"); (iii) railway, aeroplane, tram, boat, taxi-cab and cab fares or the cost of hiring a vehicle for conveyance to or returning from a professional visit.

In cases where a motor-car is used by a medical practitioner for both professional and private purposes and no attempt has been made by the practitioner to dissect his motor-car expenses, an arbitrary apportionment should be made. Since the present petrol restriction regulations have been in force the Commissioner has been allowing the taxpayer to claim nine-tenths of the expenses as being attributable to professional purposes.

This proportion, however, may be varied where the practitioner practises mainly in consulting rooms or where little or no car travelling is necessary.

**Stationery, Stamps *et cetera* (Item Number 65).**—The cost of all printing, stationery, stamps and telegrams incurred in connexion with the practice will be allowed as a deduction.

**Gas, Electricity and Telephone (Item Number 65).**—A proportion of the cost of gas or electricity or other means of lighting as would refer to that used in connexion with

the medical practice may be claimed as a deduction together with the amount paid for telephone during the year.

**Premium Paid in Respect of Lease (Item Number 66).**—Any sum paid by a taxpayer by way of a premium, fine or foregift to any person for or in connexion with the grant or assignment of a lease is allowable as a deduction from income, spread over the unexpired period of the lease; provided that the lease is for a definite term in excess of twenty-four months as distinct from a weekly tenancy or a lease from year to year.

**Other Business Expenses (Item Number 70).**—Any other expenses, not elsewhere included, necessarily incurred in the production of income should be shown under this heading, for example: (a) cost of bandages (when no dispensary is kept), (b) cost of chemicals (when no dispensary is kept), (c) accountancy fees, (d) bank charges, cheque books *et cetera*.

### Concessional Allowances.

Statement Number 9 on page 4 of the return sets out clearly the various items in respect of which a taxpayer can claim for concessional allowances subject to rebate of tax.

It will be noted that a rebate is allowable in respect of the following classes of dependants, provided that the dependant is a resident of Australia and is wholly maintained by the taxpayer.

(NOTE.—A spouse, female relative, not being the taxpayer's mother, or daughter-housekeeper, is deemed to be wholly maintained if her separate net income for the year does not exceed £100, but a proportionate rebate only may be allowed if the separate net income exceeds £50, but does not exceed £100, or if person is wholly maintained (or, in the case of a housekeeper, is wholly engaged in keeping house for the taxpayer) for part only of the year. A mother in receipt of income, including any pension, is not wholly maintained.)

(a) Spouse.

(b) Mother.

(c) Female relative of a widowed person, having the care of any of the taxpayer's children under sixteen years of age.

(d) A housekeeper of a widowed person (if wholly engaged) having the care of any of the taxpayer's children under sixteen years of age (not subject to separate income condition).

(e) A daughter-housekeeper of a widowed person.

(f) Children under sixteen years of age at July 1, 1946 (not necessarily resident in Australia), and each invalid child of the taxpayer over sixteen years of age at that date.

(g) Children over sixteen years of age at July 1, 1946, receiving full-time education at a school or university.

The following items of domestic expenditure paid in respect of taxpayer, spouse or children under twenty-one years of age are subject to rebate of tax:

(a) Medical expenses paid to a duly qualified medical practitioner, nurse, chemist or public or private hospital on account of illness.

(b) Dental expenses paid to any legally qualified dentist for dental services or treatment.

(c) Optical expenses paid to any legally qualified person for testing of eyes or prescribing of spectacles or to any person for the supply of spectacles in accordance with such prescription.

(d) Remuneration of attendant for services arising out of blindness or permanent confinement to a bed or chair.

(The maximum amount on which rebate is allowable in respect of (a), (b), (c) and (d) above is £50, including up to £10 for dental expenses.)

(e) Artificial limb (or part of limb), artificial eye or hearing aid.

(f) Funeral expenses, not exceeding £20 paid in respect of taxpayer's spouse or children under twenty-one years of age.

The following items are also subject to rebate of tax:

(a) Life assurance premiums paid, and payments made to a friendly society, superannuation fund, sustenance, or similar fund (maximum amount allowable £100).

(b) Rates and land taxes, for which the taxpayer is personally liable, paid on properties from which no income is derived, not including amounts paid for excess water.

(c) Gifts of £1 and upwards to public charitable institutions and public universities in Australia, to public funds for the relief of persons in Australia in necessitous circumstances, to patriotic funds and to the Commonwealth, or State, when made for purposes of defence, and public authorities for research respecting disease, and to approved research institutes for scientific research *et cetera*.

(d) Calls paid to mining companies and mining syndicates (operating in Australia), mining for gold, silver, base metals, rare minerals or oil, and companies carrying on afforestation as their principal business in Australia are subject to

rebate of tax and a list should be attached showing details of any such payments.

#### Additional Transactions.

I have enumerated above the various items of income most generally received and expenditure incurred by a medical practitioner in carrying on his professional practice. Several other items of income and expenditure *et cetera* may be mentioned which may not necessarily affect a medical practitioner's return in so far as his professional transactions are concerned; it should be specially noted, however, that the following are liable to taxation or may be claimed as deductions.

#### Income from Property.

**Rents** (Item Number 16).—The gross amount of rent received from properties, including rent from subletting, must be shown alongside this heading. A detailed list giving the source of such revenue must accompany the return.

**Dividends** (Item Number 17).—The gross amount of all dividends from companies, including shares distributed as bonus shares, must be shown under the heading of dividends.

**Interest** (Item Number 18).—Particulars of all interest received from mortgages, deposits of all kinds, savings bank deposits, also from bonds, stocks or debentures issued by companies or public bodies and by a State Government or by any State authority after December 31, 1923, and from all taxable Commonwealth loans must be shown in detail on statement embodied in the return.

#### Deductions from Property Income.

All expenses actually incurred in gaining or producing assessable income from property are allowed as a deduction and should be shown under the following headings on page 1 of the return.

**Rates and Taxes** (Item Number 23).—The total amount paid during the year of income for water, sewerage and drainage rates and State and Federal land tax.

**Repairs** (Item Number 24).—All expenditure on repairs and maintenance (not including alterations, additions or improvements) to properties from which rent is received.

**Insurance** (Item Number 25).—Amounts paid for insurance premiums (fire, burglary *et cetera*) on income producing property.

**Interest** (Item Number 26).—Interest actually paid on money borrowed for the purpose of acquiring and/or maintaining the property.

**Commission** (Item Number 27).—Commission paid for collection of income from property.

**Rent** (Item Number 28).—Any rent paid for property, or part thereof, from which rent is received, will be shown under this heading.

The above-mentioned deductions (in some cases similar to those previously mentioned in this article) apply only to that portion of such expenditure which refers to income received from property, and will be claimed under this section of the return by taxpayers having income-producing investments outside their professional practice.

#### Sale of Goodwill.

Under the provisions of the *Income Tax Assessment Act*, 1936-1947, the taxable income of a taxpayer shall include any premium or consideration received for or in connexion with any goodwill attached to or connected with land, a lease of which is granted, assigned or surrendered.

Consideration received in respect of the sale of goodwill of a professional practice carried on on freehold property is not assessable where the freehold is sold.

Where goodwill attaches to a lease of land or a tenancy the proceeds of the goodwill will be assessable as income attributable to the grant or assignment of the lease or tenanted premises.

It may be pointed out, however, that the courts have decided that goodwill may be either "locational", that is, attached to the situation or location of the premises, or "personal", that is, attaching to the skill or reputation of the owner.

Locational goodwill, being attached to the land, is correctly assessable as taxable income.

Personal goodwill not in any way attached to land or premises cannot be deemed to be assessable income.

I would specially point out, however, that many difficulties arise in determining on general terms whether, under present-day conditions, consideration received in respect of the sale of goodwill of a professional practice is entirely attributable to the personal skill of the vendor or is in any way associated with leased premises or its location.

Each individual case must be treated entirely on its own merits, and it is therefore advisable for a taxpayer to seek professional advice on this subject.

#### Expenses Not Allowed as Deductions.

The following items of expenditure are not in any circumstances allowed as deductions from income, except where specially mentioned:

(a) Additions or alterations (as distinct from repairs) to trade or other income-earning premises.

(b) Additions to plant and machinery (except in mining businesses under certain conditions).

(c) Expenditure incurred by a taxpayer, in the year of income, in or for the maintenance of his wife or any member of his family under the age of sixteen years, whether or not the expenditure was incurred in the production of assessable income.

No deduction will be allowed of any payments made by a taxpayer to a relative unless the Commissioner is satisfied that the payments are reasonable in amount, and *bona fide* made in the production of assessable income.

(d) Cost of travelling between taxpayer's private residence and place of business.

(e) Doubtful debts.

(f) Expenditure incurred to protect income.

(g) Federal income tax.

(h) Insurance (fire or burglary) on household furniture or personal effects.

(i) Interest not actually incurred in the year covered by the return.

(j) Interest paid on money which is not used to produce income.

(k) Losses not connected with or arising out of the taxpayer's trade or business.

(l) Purchase money paid, except for trading stock.

(m) Rent of private residence.

(n) Repayment of moneys borrowed, including amounts of principal included in annual payments on loans.

(o) Wages to persons not employed in the trade or business.

(p) Repairs, insurance, interest paid and other expenses incurred in connexion with the taxpayer's private residence. (Rates and taxes are subject to a concessional rebate.)

#### Preparation of Return.

The income tax forms supplied by the taxation departments in each State of the Commonwealth are similar in set-up. A medical practitioner practising his profession on his own behalf or in partnership will use form "B" (Business and Professional Return), which is divided into four parts marked "A", "B", "C" and "D".

Income and allowable deductions of a professional practice should be entered against the appropriate headings (referred to above) in part "C" on page 2 of the return. The surplus of total income over total deductions (net income) is then entered opposite item number 8 in part "A" on page 1 of the return.

When the return has been completed to the extent referred to above, all transactions in connexion with the professional practice should appear thereon.

It will now be necessary in every instance where a medical practitioner is in receipt of any income from investments outside his medical practice to enter all such income and allowable deductions alongside the appropriate items numbered 16 to 29 inclusive in part "B" on page 1 of the return.

Part "A" should now be added so as to show the total income and total deductions: the difference being the net income from personal exertion shown opposite item number 15.

Part "B" will be similarly added showing net income from property opposite item number 30.

Items number 15 and number 30 will now be added together to show the total net income from personal exertion and property.

Finally, the return and all accompanying schedules, if any, should be signed personally by the taxpayer, when possible, and lodged with the taxation department in the State in which the income is earned on or before August 31, 1947, unless an extension of time in which to lodge the return has been applied for and granted by the taxation department. Returns furnished on form "A" and form "S" should be lodged on or before July 31, 1947.

#### Verification of Concessional Claims.

All claims for rebate of tax in respect of donations and gifts to charitable institutions must be supported by receipts, cheque butts or some other evidence of payment.

Receipts for medical, dental and optical expenses *et cetera* should not be forwarded to the department with the return, but must be retained by the taxpayer and produced to the department on request.

#### Statements on Return.

The return contains several statements numbered 1 to 9 inclusive, which must be filled in by the taxpayer, if applicable to the transactions of his practice and if any deductions or concessional allowances are claimed in respect of the headings printed thereon, namely:

**Number 1 Statement.**—Details of salaries, wages *et cetera* paid or allowed to each employee who is a relative or is employed partly in the private domestic establishment of the taxpayer.

**Number 2 Statement (Zone Allowance).**—If a special deduction is claimed for zone allowance, the place of residence within zone, nearest post office and period of residence should be shown in this statement.

Zone allowance is a special deduction to persons residing in isolated areas. Zone A covers portions of Northern Territory, Western Australia and Queensland, while some other areas of these States, together with parts of New South Wales, South Australia and Tasmania, form Zone B. No part of Victoria falls within either of the prescribed zones. A copy of the map delineating the respective zones may be inspected at any post office within the zones or at the taxation offices.

**Number 3 and Number 4 Statements.**—These statements are not applicable to the ordinary transactions of a medical practitioner, but should they apply in any particular instance the necessary information must be supplied.

**Number 5 Statement.**—This statement will be used only on a "partnership return" furnished by two or more medical practitioners practising in partnership and calls for the names, addresses and share of each partner in the net income of the partnership.

**Number 6 Statement.**—This statement will be completed only by a medical practitioner who, in addition to the income from his professional practice, has, during the year ended June 30, 1947, received a salary from which income tax instalments have been deducted by his employer.

**Number 7 Statement.**—All dividends (including tax free dividends) received from Australian and ex-Australian companies must be shown in this statement. An appropriate note should be inserted against any tax free dividends. The total of this statement should be entered against item number 17 in part "B" of the return.

**Number 8 Statement.**—Particulars of Australian and ex-Australian interest to be shown in this statement. Special care should be exercised in supplying all the details asked for under the heading of "Interest on Commonwealth Government Loans", as those issued after January 1, 1940, are subject to a rebate of two shillings in the pound on the amount of interest received.

The total of this statement should be entered against item number 18 on part "B" of the return.

**Number 9 Statement.**—This statement refers to claims for concessional allowances subject to rebate of tax and has been referred to above under the heading "Concessional Allowances". Special attention should be given to the various headings in this statement and the several explanations therein noted, as rebates will not be allowed unless full particulars and answers are inserted as required under the various headings.

#### Detailed Lists Required.

Wherever deductions are claimed on account of any items in respect of which the form calls for a detailed list, such list must be attached to the return, otherwise the deductions may be disallowed.

Compliance with the above direction may also save the taxpayer much correspondence and irritation.

Separate detailed lists of the following deductions are necessary: gifts, charitable contributions *et cetera*; calls in mining companies; fire and burglary insurance premiums; medical expenses *et cetera*; rates and taxes; repairs; depreciation (items, values and rates of depreciation and manner in which same is calculated); bad debts (written off), giving name, date incurred and amount; travelling expenses; other business expenses.

#### Copy of Return.

A copy of the return, together with books, accounts, memoranda and all data from which the return has been compiled, should be kept for future reference.

#### General.

In conclusion, it might be pointed out that the above explanations and directions have been confined to the general transactions of a medical practitioner in the ordinary course of his practice; they have been supplemented by a few remarks regarding concessional deductions together with certain items of income from property, which may possibly affect the return.

Many fine legal points that may arise in such rare instances, as when extraneous transactions have occurred, have not been dealt with (in such cases special inquiry should be made at the department of taxation in the respective States). Apart from these I believe that should the above directions be carefully followed, the full amount of deductions as allowed by the *Income Tax Assessment Act* will be obtained.

ROBERT J. STIFFE, F.C.A. (Aust.).

Sydney,  
July 28, 1947.

## British Medical Association News.

### SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on May 29, 1947, at the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, Dr. H. R. R. GRIEVE, the President, in the chair.

#### Criminal Responsibility.

DR. W. E. AUDLEY read a paper entitled "Criminal Responsibility" (see page 175).

DR. E. P. DARK read a paper entitled "Criminal Responsibility" (see page 176).

PROFESSOR W. S. DAWSON, in opening the discussion, remarked that as a criminal, which he understood from Dr. Dark was merely another term for an ordinary member of society, he thought that some consideration should be paid to the viewpoints on the question of mental states and responsibility. Through the ages and more recently during the last century there was evidence of increasing recognition in certain circles of the mental factor in law breaking. It was just over one hundred years ago that the famous McNaughten rules had been pronounced, and these were still being discussed, although in actual practice they were very seldom followed. They were, however, in Professor Dawson's opinion, a good basis for further argument, even though medical opinion quite frequently adopted a much wider view. In regard to the certifiably insane, Professor Dawson suggested that they should never be considered responsible for their actions, and in actual practice that was the principle adopted even when a sentence had been passed. Professor Dawson pointed out that frequently the Home Secretary in the Home Country and the corresponding authority in Australia ordered an examination after trial and humanitarian principles were invariably adopted and the certifiable individual was committed to an appropriate institution. He thought that the question that should be discussed was whether the certifiably insane person might also be considered fit to plead in his own defence. The mind was now recognized as a whole, and reason and emotion were no longer divorced in the medical conception as they still appeared to be in the legal mind, and therefore it would seem that certifiable insanity might very well include unfitness to plead. Another question which was important was whether it would be fair to try a man in his absence, however desirable it might be to establish the fact that he had committed the crime of which he was accused in the interests of justice, if he was not fit to plead. It appeared to Professor Dawson that Dr. Audley had discussed a number of matters of procedure which were obviously debatable and about which there was no set policy in the official mind, and Professor Dawson suggested that perhaps the New South Wales Branch of the British Medical Association might call a committee and invite members of the legal profession to join with a view to making alterations on current procedure and possibly some improvements. Dr. Audley had mentioned certain classes and had used the term "some others", and Professor Dawson thought that these "others" constituted by far the greatest problem, as they would include those individuals who are not certifiably insane or grossly subnormal and might be above average intelligence, but rather temperamentally unstable, and could be proved to be what were known as psychopaths and the



severer types of psychoneurotics. This class would also include many of the sex offenders. Some allowance had also to be made for partial degree of responsibility as Dr. Audley had suggested, but although such persons would not be considered irresponsible—according to the McNaughten rules—it was perfectly sound on medical grounds to try to indicate what partial degree of responsibility or irresponsibility they might display, and it would then be left to the discretion of the court to award a sentence and to recommend whether or not some special care should be provided. Legal procedures in the past had been very rough and ready and, it would be remembered, the original attitude was one of *Lex Talionis*, and from that had grown a more humanitarian aspect. Punishment, deterrence and reform or rehabilitation were three objects for the passing of sentences by the court. It would appear that in the third of these objects great developments were desirable. They knew and heard frequently of the man who was constantly in and out of trouble. Professor Dawson suggested that if the opinions of psychiatrists were asked for more frequently about such cases, it would emerge that a longer period of detention than was commonly awarded by the court would be recommended. It was found, however, that when an opinion was sought it was in the hope that the psychiatrist would suggest a more lenient or more pleasant period of detention than that which was likely to be awarded by the court. This was unfortunate, and Professor Dawson thought that Dr. Dark would agree that members of the medical profession were not doing their duty to society or to the individual law-breaker by taking such a short-sighted view. He also thought that there should be more determination and more highly organized attempts at rehabilitation, but this could be done only by the provision of highly trained staffs who could devote their whole time to this particular object. Professor Dawson remarked that it would appear from at least one of Dr. Dark's examples that the clever criminal "got away with it". This was a point which was often forgotten, and in surveys that had been made on prison inmates it had generally been pointed out that there was a proportion, a high proportion, of the emotionally unstable and the more or less defective. In fact it was the poor, inefficient, sometimes physically as well as mentally subnormal, type of individual that got caught. The clever criminal frequently "got away with it". Professor Dawson said that he had recently referred to Mercier, the great English authority on the subject of crime and responsibility and mental disorder, and Mercier had pointed out that some years ago in English statistics only one-sixth of the crimes committed were ever brought to court; the inference was that the criminal one-sixth comprised the subnormal who were not clever enough to get away and to avoid detection and that fact would demand a more humanitarian aspect being shown towards the particular class who were caught. It was evident that the present methods were inefficient and more should be done either in the way of rehabilitation or further protection of society against that class who repeated their crimes after varying terms of punishment. The subject was a vast one, and Professor Dawson had been able to touch on only a very few points that had been raised. He thanked the speakers and expressed indebtedness to them for the way in which they had dealt with the subject.

Dr. GUY GRIFFITHS said that he was astounded at the different points of view put forward by Dr. Audley and Dr. Dark. Dr. Audley had assumed that the subject was one of criminal responsibility of the accused, while Dr. Dark had taken a much wider view and almost put aside the responsibility of the person who had committed the crime and frankly and boldly accused society as a whole as being responsible for the commission, not of any particular crime, but of all crime. There was a great deal to be said for such a viewpoint, and Dr. Griffiths was sure that Dr. Dark was perfectly correct in his assumption that brutal punishment increased brutal crimes and that flogging almost invariably did more harm than good. Many boys after being flogged had been turned into sexual perverts or had become sexually unrestrained, their passions having been excited by the flogging itself. Dr. Griffiths said that he was not aware that the increase in the price of bread and the increase of crime had been definitely shown to be parallel, but was quite willing to accept Dr. Dark's statement. He certainly agreed that the exhibition of sexual films from Hollywood was a serious contributor to crime in New South Wales as elsewhere. In regard to Dr. Dark's statement about criminals being made and not born, Dr. Griffiths thought that some of them were born. However, he agreed that environment played an important part and acted in a double way. First of all environment moulded character and so character, of a kind unable to resist temptation to crime, developed. Later on the environment offered the suggestion for a particular crime and the crime was then the inevitable

result of the temptation acting on the person of weak character, the weakness being due partly to innate constitution and partly to bad training in youth. If all these points were taken together it would appear that Dr. Dark took the deterministic view, a view that everything a man did was the inevitable result of his original constitution, of his up-bringing and of the particular circumstances in which he found himself when he did a particular act, criminal or otherwise. If a man's actions were the inevitable outcome of his nature and of the particular circumstances operating at the time, how could it possibly be right to hold him responsible? Dr. Griffiths was of the opinion that Dr. Dark did not go quite far enough in his views in regard to holding the criminal responsible. The deterministic view was one he did not follow in that respect completely; but Dr. Griffiths thought that the criminal had to be held responsible because it was the duty of society to protect itself. As Dr. Dark had implied and Professor Dawson agreed, it was very wrong for society to revenge itself on the criminal, because retaliation was wrong and punishment useless, unless corrective, as it might be in some cases. It certainly did not succeed in all cases, as for instance the case of Moss which Dr. Dark had detailed, but correction did succeed in some measure. As Dr. Dark had suggested, it would succeed in many more cases where it applied at an earlier stage—when the criminal was quite young. If he had been brought up in a good environment, praised for his good deeds and blamed for his wrong deeds, his character would have developed in such a way that he would be able to resist temptation when it came and he would be much less liable to lapse into crime. Dr. Griffiths stressed the view that punishment should never be vindictive, that there should be no retaliation. Correction should be applied not only after conviction of the crime but from early infancy so that it would mould character. Punishment, inflicted as an example to prevent others, might fail completely because crimes were committed at a time when the criminal could not carry in his mind any recollection of past example. If the deterministic theory of conduct held that punishment should never be vindictive, but should be applied early in infancy, it was useless to employ it as an example to others. There remained therefore only the fourth reason for punishment, and this was the protection of society. It was well known that terrible misfortunes had happened because society was not protected from a particular criminal when it had been known he was dangerous. In Dr. Griffiths's opinion it was wrong to go so far to the other extreme and to allow criminals to escape by leaving the country unpunished to carry on their activities in other parts of the world as it was wrong in the other direction to inflict harsh, cruel, vindictive punishment. Dr. Griffiths thought that everyone would agree that Dr. Dark had made a very strong case when he emphasized the view that many criminals became criminals through poverty-stricken environment as children. Dr. Griffiths, however, could not agree with Dr. Dark that all criminal responsibility rested on the community at large.

Dr. D. W. H. ARNOTT said that he had noted that when serious offences were being tried in court and the question of responsibility arose, the judge always insisted that it be viewed within the strict limits of the McNaughten rules. Dr. Arnott said that Mr. Justice Stephens, in his "History of the Criminal Law of England", was in favour of some relaxation of the McNaughten rules and took the sensible view that a delusion of any kind, even if it could not be linked with the particular crime, indicated such a gross disorder of mind that a sound judgement as to rightness or wrongness would be impossible. The concept of partial responsibility was unreal and shadowy and most psychiatrists would be glad to avoid it; nevertheless where actual mental disease or deficiency existed in association with the committing of a crime there should be a widening of the McNaughten rules.

In regard to Dr. Dark's paper, Dr. Arnott said that he could not agree with a great deal of it, but there was no doubt that character was determined largely by early environmental influences, but just what were the best influences was highly speculative. Aggressiveness was a fundamental human quality and had to be given proper expression, and if not satisfied in a constructive way often burst forth in an anti-social way. Whether there was going to be less frustration in a completely socialized State, Dr. Arnott did not know, but he feared there would be more. He had worked for the Government and for the army, and from his experience he would not choose socialization after seeing the mass frustration and consequent mental ill health which took place in the army. Private individuals might perpetrate aggressive crimes against their fellow men, but at least the law provided some control and redress, but the repressive mass aggression which a socialized State could

impose upon its helpless citizens admitted of no escape and of no redress.

Dr. GREY L. EWAN said that in view of the modern development in the understanding of mental disorder, all forensic psychiatrists agreed that the McNaughten criteria were not a satisfactory standard for the administration of the criminal law in the present-day courts of law. However, the interpretation given to the McNaughten rules was becoming more liberal as time passed. All psychiatrists knew that some patients in mental hospitals were dangerous and some were obviously insane. Paranoia was a rare form of mental malady in which systematized delusions were present, concentrated on one particular aspect of the patient's life situation with which he was solely concerned; in all other respects one would hardly know that such a patient was alienated at all. He could comply with the legal criteria of fitness to plead; he knew the nature and quality of his act, he could instruct his counsel and he could follow his trial intelligently, but he was none the less very dangerous. Dr. Ewan went on to say that Dr. Audley had put forward a proposition that the fact of guilt should first be established, and that during the trial the defending counsel should not be allowed to raise the question of insanity in his defence. Dr. Ewan thought that they would never succeed in having that provision made; in any case, it would be a dangerous principle. The proposal raised a conflict between the medical opinion of unsoundness of mind and responsibility and the legal viewpoint. As Professor Dawson had pointed out, this conflict arising out of the commission of crime was due to a divorcing of the emotions from the reason or intellect, and in the synthesis or integration of the personality as a whole that could not be done. That was where the difference arose between the legal and the psychiatric concepts. Dr. Ewan went on to say that Dr. Dark had raised the question of the influence of heredity and environment on crime. A great deal of ink had been spilt on that subject. Dr. Ewan had had much experience in examining criminals in the State Penitentiary, and he was becoming more and more convinced that some criminals were born and not made. If one was confronted with psychiatric evidence of perverted instinct, it was clear that the instinct had been perverted from childhood, and such persons became a menace to society. Dr. Ewan did not mean to say that environment did not have much to do with the question. But psychiatrists had to take the middle road. Each case required a very careful analysis and a very careful anamnesis to ascertain exactly what degree of responsibility existed in law if expert evidence was to be given in court. The question of culpability was wrapped up with the question of responsibility, and it was difficult to separate the two. Sometimes one had to decide on what degree of culpability existed and which might be regarded or taken into account as mitigating circumstances when the punishment was being determined. As psychiatrists they were entitled to discuss the merits and demerits of degrees of punishments; but the statutory authority had the inflicting of punishments, and it looked to the psychiatrist to assess the degree of the accused's culpability. The present tendency was for a certain amount of morbid sentimentality to surround the interests of these criminals. Psychiatrists had to be careful in making reports that they were not influenced by this morbid sentimentality in the public mind; they had to be fair in their judgements and not influenced in any case in which they believed that the accused person knew the nature and quality of his acts. They might have their own individual psychiatric viewpoint; they might consider the accused, who was legally responsible in the terms of the McNaughten rules, to be mentally unbalanced. No doubt they could express an opinion; that was where culpability entered the matter. In conclusion, Dr. Ewan again drew attention to the existing tendency not to weigh carefully the respective degrees of responsibility and culpability.

Dr. C. H. JAEDE said that one thing which was of great interest to him in discussions between the psychiatrist and the medical sociologist was the question of punishment and the effect of punishment on the person who had committed a crime, whether he was sane or not. Dr. Jaede had two friends who were judges. Both of them, when confronted with the responsibility of sentencing pervers or the committing criminal (the petty thief, for example) or the person who had been considered guilty of assault or personal damage with intent, found themselves in great difficulty in determining what sentence to give. In the case of the pervers, they were apparently limited by the law, which stated that these persons must be sent to prison or allowed to go free. The judges themselves knew that the greater proportion of pervers were mentally sick and had some kink in their characters which did not necessarily make

them unfit for society. In certain cases they might be perfectly unfit for society. Some were men and women of keen intelligence. It did not appear to these judges to be correct to send them to the ordinary gaol for the ordinary type of punishment. With regard to the thief, they were in difficulty again. The two concerned had two different methods. One believed in sending the prisoner to gaol. The other believed after investigating such cases that the greater proportion of these persons committed these crimes as a result of poor up-bringing and poor circumstances, and that he must try to make better citizens of them. The great majority he released on bond. He believed that if he sent them to prison they would become criminals because of their constant association with criminals. In the case of criminals found guilty of bodily assault, one judge believed in savage sentences, the other in not such savage sentences. Dr. Jaede said that he had been interested in the statement that if flogging was instituted it would make the victim worse. But, as he had shown, the judiciary had different conceptions of punishment and of what was correct punishment, and in certain cases they were bound by law to give punishments which they considered not correct. Dr. Jaede said it was interesting, when one discussed the matter with policemen, to find that they were particularly keen to see criminals guilty of bodily assault severely punished. In fact, the police were rather disgusted if such criminals were let out on bail. The point he was reaching was that surely at this stage three classes of people needed some educating by those who could appreciate the effects of incorrect punishment; the people concerned were the Government, the police and possibly the medical profession. Possibly the members of the medical profession should accept their responsibility and endeavour to influence both the other bodies so that society would be somewhat guarded by means of a correct attitude towards these various sentences. The main point was that he felt that certain methods of incarceration produced criminals, and it should be the task of the medical profession to see that these criminals were rather made better members of society.

Dr. Grieve, from the chair, thanked the two speakers for their interesting papers, and all those who had taken part efficiently and interestingly in the discussion. Dr. Grieve said that he had been glad to hear the suggestion from Professor Dawson that some steps might be taken by the New South Wales Branch Council to bring about consultation between representative members of the medical profession interested in the question of criminal responsibility and representative lawyers. The suggestion was worth following up. Dr. Grieve asked Professor Dawson to consider in the near future elaborating the suggestion in a communication to the Branch Council. Very shortly it was hoped to have in being in New South Wales a medico-legal society, which would perhaps help in solving some of these problems. Already consultations had taken place between the Council and representatives of the Bar Council and of the Law Institute, and a working constitution had been drawn up for the society; it was hoped that within a month or so the inaugural meeting of the society would take place. Dr. Grieve went on to say that the questions raised by the two speakers prompted him to make one or two observations. He had been concerned to hear Dr. Audley suggest that a person charged might be both certifiable and fit to plead. Dr. Ewan had pointed out the danger that would arise in a certain eventuality in this case; an example would be that such a person, having been tried, and having been disqualified from raising the plea of insanity, might still be convicted of a crime of which on medical grounds he knew not the nature or the consequences. Dr. Grieve was concerned with the possibility that such a person might be deemed fit to plead and allowed to plead insanity, and that such a plea of insanity might be either accepted or rejected by a jury. In either case the consequences might be dangerous to the criminal or to the public at large. On the whole it seemed that this was a suggestion that should be considered rather more fully. Referring to environmental influences towards crime, on which Dr. Dark had insisted, Dr. Grieve said that he felt that they were in danger of accepting too narrow a conception of the meaning of environment. They were apt to think of environment in terms of financial status in society, when, as they all knew, environment was a vast and complex thing. Some little time previously he had seen the case histories of a fairly large number of male pervers, who would all have been guilty of a crime according to the accepted code of law. He had been struck with the similarity of the life histories of all these people. They came from all strata of society. There was no predominance of origin in one group of society or another. Many came from good homes; in fact, none of them stated that they had been brought up in poor circumstances. Many of them



had been brought up in the country and many in the city. But one thing was common to them all, that they had all been either only sons or only children, or they had been pampered by their mothers. The mothers had taken a peculiarly close interest in them. At a very early age, in some cases beginning at five years, in no case later than seven years, they had resorted either alone or in company to some form of perversion. That fact seemed to indicate that the environmental influence was not the only one, and that it was certainly not a narrow one. Dr. Grieve had also been interested to hear Dr. Arnott refer to the common quality of aggressiveness in young people. Some years earlier Professor Thrasher had made a personal research into 1,100 of the 1,300 gangs in Chicago, and certain general conclusions arose out of his work; they were very general and were confirmed in analysis of the life, creation and character of every gang. The chief conclusion was that gangs were composed almost entirely of young men and boys who showed in a pronounced degree this quality of aggressiveness. Out of that Professor Thrasher suggested the formation of some association or club or community meeting place where an outlet would be provided for that aggressiveness in games, boxing and other types of contest. Dr. Grieve said that brought him to the excellent work done by the Police Boys' Clubs in New South Wales. Only 5% of boys who were members of those clubs fell later on into the hands of the police. Another striking thing was that those boys frequently did not move up economically except by their own efforts; they remained in the same districts and lived under the same conditions, which might be crowded and confined. In spite of that fact—that their environment in the narrow sense of the word remained unchanged—90% of the boys grew up free from the commission of crime. The only inference seemed that when thinking of environment one should not have too narrow ideas—that one should consider all the activities and factors that made up the environment.

Dr. Audley, in reply, said that his paper had necessarily been a short account, as it had had to be confined to twenty minutes. The subject was tremendously wide, and he had deliberately chosen a narrow section of it. He had wished to provoke discussion, and had been happy when the discussion took place. Dr. Ewan had mentioned an important matter when he raised the two questions of responsibility and culpability; he (Dr. Audley) had wanted the question of culpability to be dealt with separately from that of responsibility. The point was that it should be determined whether a particular crime had been committed by a particular person, so that the position would be avoided in which a person was sent to a criminal mental hospital and left there for ten years without its being determined whether he committed the crime or not. Advantage should be taken of the present provisions of the law, that fitness to plead was something less than insanity. The question of insanity should be allowed before and after trial, but not during trial. The question of fitness to plead should be determined in the first instance. If a person was not fit to plead, then he would have to be placed in a mental hospital. If he was fit to plead, but insane, it was better to give him a chance to plead. But when the verdict was given, the judge should have a great deal of discretion in awarding the sentence, and should not be forced to sentence a murderer to death. Dr. Audley said that he would like to see the death penalty go. That would overcome some of the difficulties about morbid sentimentality existing at the present time. These punishments were bad for the community, and the newspapers dealt with them in a horrible fashion that was conducive to more crime. If the plea of insanity could not be raised during the trial, but only afterwards, then the judge could have an opportunity to take a psychiatrist's opinion about it. There should be a psychiatrist attached to the court, to watch every case in which the question of insanity arose and to observe the criminal frequently and constantly. If advantage was not taken by the defending counsel of the provision concerning the question of fitness to plead, he could raise it later, on the question of sentence, and the judge could send the prisoner, not necessarily to prison, but to some other institution. Dr. Audley believed that in the future there would be other institutions than criminal mental hospitals and gaols. He had been interested in the proposal that a conference on this question should be held with the legal profession to determine the principles; some modification of the McNaughten rules seemed necessary. Dr. Audley said that those were the main points, and he hoped that what he had said had made his position clear. Another question from the sociological point of view was that of parental education. It was certain that the childhood training of those who were insane and of those who committed crimes was defective. To deal with this it was necessary to have a long-range policy of parental

education, and in the meantime criminals had to be dealt with. It was impossible to get away from the concept of partial responsibility. There were the sane and the insane, and there were persons who were not certifiably insane and yet not quite normal. That provided the question of partial responsibility. If the question of insanity was raised before sentence was given, as he had suggested, then the judge would have an opportunity to detain these people for as long periods as necessary. Dr. Audley did not like the idea that a man sentenced to twenty years' imprisonment in gaol for murder might be released in fifteen years, unless there was no danger of his doing the same thing again. Many people were morbid. If these things were dealt with in court, and if judges continued to be regarded as impartial, better justice would be done in the future than was being done at the present time.

Dr. Dark, in reply, said that various important points had been raised. The first was Professor Dawson's question about terms of imprisonment. Dr. Dark said that it seemed to him that in dealing with a criminal only two objects were of any importance: (i) to protect society, (ii) to rehabilitate the prisoner. A short term of imprisonment probably served neither of these. Dr. Dark remembered a case some years previously in Victoria. A man was convicted of a violent sex crime. He was given a number of strokes of the lash and imprisoned for two years. He left prison after having served his sentence, and very soon he committed a much worse sex crime. That sentence had obviously done nothing to rehabilitate him or to protect society. That linked up with a remark made by Dr. Jaede, that judges were compelled to give certain sentences for certain crimes, and that obviously was correct in the present form of the law. Dr. Dark thought that a law was obviously bad when it did not fulfil its task of protecting society and rehabilitating the criminal. All intelligent members of society should try to have it altered. That was a matter in which the Association should carry some weight, and if some consultation with the lawyers could be arranged, it would be a step towards making society a better place to live in. In reply to Dr. Griffiths, Dr. Dark said that he thought that he (Dr. Griffiths) believed him (Dr. Dark) to have insisted that all criminals were made by society. In dealing with a subject so enormous as the responsibility of society, one could not do more in twenty minutes than sketch out salient points. Some people must obviously be born with a tendency to crime; everyone was born with some tendencies, ranging from musical genius to crime. But even then, society played some part in shaping their destiny. If they were born in a society completely wise, they would be "spotted" early, and either put in an environment where the tendency was completely limited or permanently controlled so that they would not be a menace to society. The law as it existed at present neither protected society nor reformed the criminal. Most people were now deterministic in the wider sense that every person acted more or less as he had to act, if both his original endowment and the play of society upon this endowment were taken into account. Dr. Dark said that he did not feel happy about this, because he had been brought up to believe in free will. But his reading led him to believe that the determinists were correct, and that the range of free will was not very large. Dr. Arnott had been unimpressed by any move towards socialism. Dr. Dark said that he had been careful not to use the word "socialism", and that was not the time to discuss it. But he felt obliged to utter a mild protest. Government employment in a capitalistic country did not conform with employment by the community in a cooperative society. The argument from the vices of government employment to what would occur in other circumstances was not valid. It would be very beautiful to have a whole evening in which to discuss the question. Dr. Dark went on to say that Dr. Grieve had raised an important point, and he (Dr. Dark) was in entire agreement with him; the range of environment was very much more than the amount of money one had to spend and the house in which one lived. The environment must be taken to mean the total impact of all one's surroundings upon one. Dr. Grieve had said that all the members of a group of pervers who he had investigated came from families of one child pampered by the mother; that was the really significant environment for them, not their wealth or the place where they lived. Dr. Dark said that he had just read a book by Pearson on Oscar Wilde. Pearson pointed out that at Oscar Wilde's birth his mother was passionately anxious to have a girl, and for several years (until the birth of the next child) he was brought up as if he was a girl. If he was born with certain glandular deficiencies, this fact, together with his being brought up as a girl, provided his environment. That dovetailed in with Dr. Grieve's statement about sexual pervers.



## Naval, Military and Air Force.

### APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette* of July 24, 1947.

#### ROYAL AUSTRALIAN AIR FORCE.

##### *Citizen Air Force: Medical Branch.*

Charles John Newhill Leleu, O.B.E. (1176), is appointed to a commission with the substantive rank of Flight Lieutenant and the temporary rank of Wing Commander with effect from 12th February, 1947. His name is to appear in the Air Force List issued by the authority of the Air Board next after the name of Temporary Wing Commander H. J. Melville (1171), and before the name of Temporary Wing Commander A. W. Raymond, O.B.E., M.C. (271218), and the said Charles John Newhill Leleu will take seniority as an officer in the Royal Australian Air Force accordingly.

The approval given in Executive Council Minute No. 259 of 1945 to the transfer from the Reserve to the Active List of Flight Lieutenant R. K. Smyth (257715), as notified in *Commonwealth of Australia Gazette*, Number 216, dated 8th November, 1945, is withdrawn.

##### *Reserve: Medical Branch.*

The appointment of Temporary Wing Commander C. J. N. Leleu, O.B.E. (1176), is terminated, 11th February, 1947.—(Ex. Min. No. 52—Approved 23rd July, 1947.)

## Medical Practice.

### THE MEDICAL BENEFITS FUND OF NEW SOUTH WALES.

DR. J. G. HUNTER, secretary of the Medical Benefits Fund of New South Wales, Limited, advises that it is the intention of the council of the fund to open the fund to the public on August 18, 1947.

The business management of the fund will be in the hands of the Hospitals Contribution Fund of New South Wales, M.H.C.F. House, 7, Hamilton Street, Sydney.

The rates of contribution and the benefits payable will be made available to members of the profession in New South Wales in literature which is being forwarded to them.

The council of the fund is pleased to announce that close on 1,000 members of the New South Wales Branch of the British Medical Association have become medical members and that applications will continue to be received from those desirous of becoming members.

## Correspondence.

### "EPIDEMIOLOGY IN COUNTRY PRACTICE."

SIR: May I use your columns to ask whether the doctor to whom I lent "Epidemiology in Country Practice", by Piccles, which was presented to me by Sir Humphry Rolleston, would kindly return it. I am sorry to say that I have forgotten to whom I lent this classic account of disease.

Hoping that you can do this for me.

Yours, etc.,

G. C. WILCOCKS.

143, Macquarie Street,  
Sydney,

July 21, 1947.

### THE SIGNIFICANCE OF THE GENE.

SIR: In your editorial of May 24, 1947, on "The Significance of the Gene" there are some statements on which, as one interested in theoretical biology, I should like to make some comment. In the first place, while I am prepared to admit that Dr. H. J. Muller is a very able geneticist, I am not impressed by his powers as a thinker. He says, for example,

that it is very probable "that the most primitive forms of life consisted of nothing else than a gene". It would be interesting to have his definition of a gene. Presumably he regards it as a particle located in the chromosomes of the nucleus, but it is a safe bet that he knows no more about it than that, for, according to Dobzhansky, "a knowledge of the nature of the genes as such remains as yet one of the distant goals of genetics".<sup>(1)</sup>

Then, the only real evidence regarding the origin of life—that provided by palaeontology—indicates that life began with the various invertebrate forms that appear in the Lower Cambrian. Finally, it needs to be emphasized that the theory of the gene is a deduction from the philosophical postulate of mechanistic materialism and is not an inductive generalization based on the facts of heredity. This point has been well made by E. S. Russell, who shows how the gene theory "is tainted with the same faults as the particulate theory of Weismann—the resolution of the organic unity into isolated parts or characters, the opposition of the nuclear substance as active and formative to the cytoplasm as comparatively inert material, the invention of purely abstract and fictive units or parts endowed with mysterious formative powers". And he adds, "so long as the gene is regarded as a purely hypothetical concept invented for the specific purpose of explaining certain complicated facts of inheritance, no great harm is done. But when it is treated as a real existent body all the factitious and gratuitous complications of the particulate theory tend to set in".<sup>(2)</sup>

The gene theory is the application to heredity of the particulate theory of the organism, which is as old as Democritus and was decisively refuted by Aristotle in his work "*De Generatione Animalium*". There are two main types of theory regarding the nature of the living organism: the particulate theory which makes the unit of life something smaller than the organism and explains the organism as simply a collection of such units, and the organismal theory which regards the organism as the unit and smaller particles of any kind as simply parts of this unit. In the organismal theory of heredity, the unit of heredity is not the gene or any other particle of the germ plasma, but the germ cell, which is a complete individual, possessing the same nature as its parents; and heredity is not a process by which genes "perpetuate themselves in every detail", but the process by which an organic type, for example, a cat or an ape or a man, perpetuates itself. The fact that some structural detail in the parent is reproduced exactly in the offspring is unimportant compared with the fact that parent and offspring agree in type; modern genetics concentrates its attention on the unimportant detail and ignores what for theoretical biology is the main problem. "Genetics", says J. B. S. Haldane, "can give us an explanation of why two fairly similar organisms, say a black cat and a white cat, are different. It can give us much less information as to why they are alike."<sup>(3)</sup>

In fact, as the science is at present constituted, it can give us no information at all.

Like all specialists, geneticists are liable to exaggerate the importance of their science with respect to the rest of biology. But since they are restricted to organisms belonging to the same genus or species, their studies can throw little light on the problem of the evolution of the larger groups such as orders, classes or phyla; the gene theory, of which they make so much, does not make the problem of heredity any easier to understand, for if the germ cell is simply a congeries of, say, a million genes, how is it that these genes all cooperate with astonishing regularity so as to produce, say, a cat? If this work of organization is attributed to a supreme gene, in what, unless it be its hypothetical material structure, will this hypothetical gene differ from, for example, the "entelechy" of Driesch? Finally, if all the genes of an organism are present in every cell, how can they be the cause of different cells—muscular, nervous, osseous? How is it possible for completely similar causes to produce such dissimilar effects?

Dr. Muller accepts the neo-Darwinian assumption that natural selection is the principal cause of evolutionary progress as if it were an axiom admitted by all, whereas many biologists contend that natural selection plays a minor role in evolution. Robson and Richards in their "*Variation of Animals in Nature*" subject the natural selection theory to a searching criticism and show pretty clearly that natural selection is incapable of doing all the work that the neo-Darwinians ascribe to it.

I doubt very much whether the spread of dentistry is likely to cause a progressive degeneration of human teeth, just as I doubt whether prolonged disuse is really the cause of the absence of eyes in cavernicolous fish. I wonder, too, whether there is any real palaeontological evidence that the snake is descended from a reptile possessing limbs.

Geneticists seem to have a penchant for emphasizing the sociological implications of their theories, in which a pyramid of conjecture often rests on an apex of fact. To lessen the danger of the general public and the medical profession taking their pretensions too seriously, it seems worth while to point out how the conjecture is out of all proportion to the fact. Of course, conjecture often masquerades as fact. As C. S. Lewis puts it: "Hypothesis, my dear young friend, establishes itself by a cumulative process: or, to use popular language, if you make the same guess often enough it ceases to be a guess and becomes a Scientific Fact."<sup>(6)</sup>

Yours, etc., "CRITICUS."

July 18, 1947.

#### References.

- <sup>(6)</sup> Dobzhansky: "Genetics and the Origin of Species", 1937, page 30.  
<sup>(7)</sup> E. S. Russell: "The Interpretation of Development and Heredity", 1930, page 157.  
<sup>(8)</sup> J. B. S. Haldane: "The Causes of Evolution", 1935, page 61.  
<sup>(9)</sup> C. S. Lewis: "The Pilgrim's Regress", 1935, page 36.

## Australian Medical Board Proceedings.

### TASMANIA.

THE undermentioned have been registered, pursuant to the provisions of the Medical Act, 1918, of Tasmania, as duly qualified medical practitioners:

- Gardiner, John William, M.B., B.S., 1942 (Univ. Melbourne), Launceston, Tasmania.  
 Morton, Lorimer Grant, M.B., B.S., 1931 (Univ. Melbourne), Melbourne, Victoria.  
 Chamberlain, Norman John, M.B., B.S., 1942 (Univ. Melbourne), Port Cygnet, Tasmania.

## Nominations and Elections.

THE undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

- Manning, William Kenneth, M.B., B.S., 1939 (Univ. Sydney), Repatriation General Hospital, Concord.  
 Thomson, Helen Elizabeth, M.B., B.S., 1946 (Univ. Sydney), Kiewa, Church Street, Pymble.  
 Mandel, Eva, provisional registration, 1947 (Univ. Sydney), Killiecrankie, MacLaurin Parade, Roseville.

## Medical Appointments.

Dr. J. T. Cullen has been appointed medical officer, Department of Public Health, New South Wales.

Dr. A. Fryberg, Director-General of Health and Medical Services, has been appointed Inspector of the School of Anatomy, Brisbane.

Dr. T. E. V. Hurley has been appointed a member of the Charities Board of Victoria, pursuant to the provisions of Section 9 (a) of the Hospitals and Charities Act, 1928, of Victoria.

Dr. B. P. McMenamin has been appointed relieving medical officer, Mental Hygiene Service, Department of Health and Home Affairs, pursuant to the provisions of The Public Service Acts, 1922 to 1945, of Queensland.

## Books Received.

"Textbook of Obstetrics", by Gilbert I. Strachan, M.D., F.R.C.P., F.R.C.S., F.R.C.O.G.; 1947. London: H. K. Lewis and Company, Limited. 9½" x 6½", pp. 746, with many illustrations, some of them coloured. Price: 45s.

"Elementary Physics for Medical, First Year University Science Students and General Use in Schools", by G. Stead, M.A. (Cantab.), D.Sc. (London), F.Inst.P.; Seventh Edition; 1947. London: J. and A. Churchill, Limited. 8½" x 5½", pp. 588, with many illustrations. Price: 15s.

"Current Therapies of Personality Disorders", edited by Bernard Glueck, M.D.; 1946. London: William Heinemann (Medical Books), Limited. 9" x 6", pp. 302. Price: 17s. 6d.

"Diseases of Children's Eyes", by James Hamilton Doggart, M.A., M.D. (Cantab.), F.R.C.S. (England); 1947. London: Henry Kimpton. 9½" x 6", pp. 300, with many illustrations, some of them coloured. Price: 42s.

"The Bacteriology of Spray-Dried Egg: With Particular Reference to Food Poisoning", by various authors; 1947. Medical Research Council of the Privy Council, Special Report Series Number 260. London: His Majesty's Stationery Office. 9½" x 6", pp. 66. Price: 1s.

## Diary for the Month.

- Aug. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.  
 Aug. 12.—Tasmanian Branch, B.M.A.: Ordinary Meeting.  
 Aug. 18.—Victorian Branch, B.M.A.: Finance Committee.  
 Aug. 19.—New South Wales Branch, B.M.A.: Medical Politics Committee.  
 Aug. 20.—Western Australian Branch, B.M.A.: General Meeting.  
 Aug. 21.—Victorian Branch, B.M.A.: Executive Meeting.  
 Aug. 21.—New South Wales Branch, B.M.A.: Clinical Meeting.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

**New South Wales Branch** (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phoenix Mutual Provident Society.

**Victorian Branch** (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

**Queensland Branch** (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute; Brisbane City Council (Medical Officer of Health). Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

**South Australian Branch** (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

**Western Australian Branch** (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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